A Dissertationon

A STUDY ON THE PRESENCE OF ACCESSORY MAXILLARY OSTIUM

Submitted to the

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of the degree of

M.S.BRANCH IV
(OTORHINOLARYNGOLOGY)

GOVERNMENT STANLEY MEDICAL
COLLEGE & HOSPITAL
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,
CHENNAI, TAMILNADU

APRIL 2015

DECLARATION

I, **Dr. VRINDA .B. NAIR**, solemnly declare that the dissertation, titled **"A Study On The Presence Of Accessory Maxillary Ostium"** is a bonafide work done by me during the period of January 2013 to July 2014 at Government Stanley Medical College and Hospital, Chennai under the expert supervision of **PROF. DR. T. BALASUBRAMANIAN, M.S., D.L.O.,** Professor and Head, Department Of Otorhinolaryngology, Government Stanley Medical College and hospitals, Chennai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the rules and regulations for the M.S. degree examinations in Otorhinolaryngology to be held in April 2015.

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CERTIFICATE

This is to certify that the dissertation -**"A Study On The Presence Of Accessory Maxillary Ostium"** presented BY **DR.VRINDA .B. NAIR** , is an original work done in the Department of Otorhinolaryngology, Government Stanley Medical College and Hospital, Chennai in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University for the award of degree of M.S. (Otorhinolaryngology) Branch IV, under my supervision during the academic period 2012-2015.

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ACKNOWLEDGEMENTS

I wish to express my sincere thanks to **Prof. Dr.AL. MEENAKSHI SUNDARAM, MD, DEAN**, Government Stanley Medical College and Hospital for having permitted me to utilize the facilities of the hospital for conducting this study.

My heartfelt gratitude to **Prof.Dr.T. BALASUBRAMANIAN, M.S., D.L.O.,** Professor and Head of the Department, Department of Otorhinolaryngology, Government Stanley Medical College and Hospital for his constant motivation, valuable suggestions, and expert supervision during the course of this study.

I express my whole-hearted gratitude to **Prof.Dr.N. SEETHALAKSHMI M.S.,D.L.O.,D.N.B,** Professor and Chief of ENT UNIT II of Otorhinolaryngology**, PROF. DR. F.ANTHONY IRUDHAYA RAJAN M.S.,D.L.O,** Professor, for supporting, guiding and encouraging me in this study.

I wish to thank my **Assistant Professors** DR.K.ATHIYAMAN M.S, DR.C.KARUPPASAMY M.S.,D.L.O., DR.M.P.CHANDRAMOULI M.S., DR.SARAVANA SELVAN M.S., DR.C.BHARANIDHARAN D.L.O. for their valuable tips and guidance.

I am grateful to all the other post-graduates who most willingly helped me during this study period.

I also thank the staff nurses, theatre personnel, OPD staff, Department of Otorhinolaryngology, Government Stanley Hospital for their co-operation and assistance in the conduct of this study.

I wish to extend my gratitude to my statistician for his expert assistance.

Last but not the least, I am indebted and grateful to all the **Patients and Normal volunteers** who constitute the backbone of this study, who most willingly and selflessly subjected themselves to this study for the sake of the benefit of their community and without whom this study would not have been possible.

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1. ABSTRACT

The association between accessory ostium and chronic sinusitis is still a subject of controversy as various studies have given a wide range of variable results. The prevalence of accessory ostium varies between 2 – 44 %.

Objective

To study the prevalence of Accessory ostium in those with Chronic Sinusitis and those without the disease.

Methods

83 patients with chronic sinusitis and 83 subjects without the disease were studied by nasal endoscopy for the presence of accessory ostium.

Results

30 % of those with chronic sinusitis had an accessory ostium , whereas only 10 % without the disease had it (p value <0.001). Majority of the accessory ostia were on the left and in the posterior nasal fontanelle , in both the groups. 6 (24 %) patients and 1 (13 %) unexposed subject had bilateral accessory ostia.Only 2% of

the diseased individuals had double ostia. Only 12 % showed *circular phenomenon.* However symptoms like headache , facial pain , post nasal drip and halitosis , and purulence on examination (Rhinosinusitis Task Force Criteria) were not found to have any specific association with the presence of accessory ostia.

Conclusion

To conclude we would say that the presence of an accessory ostium can be considered as an indicator of maxillary sinus disease, along with the other criteria for chronic sinusitis and that it can help surgeons in their decision making as to whether a surgery is required or not.

Key words : accessory ostium , maxillary sinusitis

2. OBJECTIVE

To study,

- The prevalence of accessory maxillary ostium in chronic sinusitis and that in normal subjects by nasal endoscopy.

INCLUSION CRITERIA:

1. Age:15-45 years.
2. Patients with clinical symptoms of chronic maxillary sinusitis.
3. Radiological diagnosis of chronic maxillary sinusitis.
4. Persistence of symptoms for more than 12 weeks even after medical line of treatment.

EXCLUSION CRITERIA:

1. <15 or>45 years of age.
2. History of previous maxillary sinus surgeries.
3. Patients with sinonasal neoplasms.
4. Patients with sinonasal encephaloceles.
5. Patients with known ciliary dysfunction such as primary ciliary dyskinesia and Kartagener Syndrome .
6. Patients with sinonasal polyposis.
7. Patients with fungal sinusitis.
8. Patients with Cystic Fibrosis .
9. Patients who are immunocompromised (with the exception of diabetics) .

3. INTRODUCTION

Chronic sinusitis is a group of disorders characterized by the inflammation of nasal and paranasal sinus mucosa which is now extremely prevalent, affecting 32 million adults , or 16.3 % of the adult population[9]. Its diagnosis is mainly based on clinical symptoms and signs, supported by diagnostic nasal endoscopy and CT scan.

Maxillary sinusitis is the side effect of evolution as a result of which man acquired an erect posture. The secretions of the maxillary sinuses have to be transported against gravity by the mucociliary action. Hence the normal functioning of a sinus depends on – patency of the ostium , normal mucociliary transport and the normal quality and quantity of secretions.

Accessory ostia also known as ***Giralde's orifice***are defects in the lateral nasal wall, in the region of fontanelles, which communicate with the maxillary sinus , in addition to the natural ostium. They are thought to develop as a consequence of damage to the fontanelles following sinus infections. This in turn becomes a reason for recurrence of infection due to mucous recirculation.

Majority of the accessory ostia occur singly. A few multiple ostia have also been observed. They are mostly acquired and rarely congenital. However there is still a controversy over its etiology.

The present study is an attempt at finding the prevalence of Accessory Ostium in those patients with Chronic Sinusitis and in those without. Here we have used Nasal Endoscopy as a tool to investigate the presence of accessory ostia. We have deliberately avoided CT PNS as a diagnostic tool as our study included normal subjects as well.

4. REVIEW OF LITERATURE

Hippocrates in the 5th century B.C itself has described sinusitis when he quoted "in a person having a painful spot in head, with intense headaches, pus or fluid running from the nose removes the disease."

Leonardo da Vinci in his period had accurately given the description of the maxillary antrum and the frontal sinus. [1]

In 1869, Wertheim made a Conchoscope to examine the anterior and middle thirds of the nasal cavity. [1]

In 1879, Nitze-Leiter developed the Cystoscope, and this marked the second stage in the developement of endoscopy. [1]

Hirschmann is considered the *Father of Endoscopy*. In 1902, Hirschmann and Valentin introduced a modified cystoscope into a maxillary sinus through an enlarged dental alveolus.[1]

During the period 1951 – 1956, Hopkins made improvements in the optics of endoscopy which included a separate light source , small diameter of the endoscope , large field of vision , excellent resolution , high contrast and constancy of color. [1]

In the 1950s Messerklinger developed a systematic diagnostic approach to the lateral wall of nose. [1]

HISTORY OF STUDIES ON MAXILLARY ACCESSORY OSTIUM

There have been various studies in the past conducted to find the presence of Accessory Maxillary Ostia (AMO) on live subjects as well as cadavers. In 1870 , *Zuckerkandl* introduced the term '*fontanelle'* for certain regions below the uncinate and above the inferior turbinate , deficient in bone covered by nasal mucous membrane medially and maxillary sinus laterally , with connective tissue sandwiched in between. *Levine et al* described the anterior and posterior fontanelle (ANF &PNF) in relation to the uncinate - PNF being posterosuperior to uncinate and ANF between the inferior edge of uncinate and attached margin of inferior turbinate[25]. Scheaffer (1953) discovered twin openings at the Hiatus Semilunaris and described them as Triple PMO. He preferred the term Duplicate PMO than Accessory ostium. Rice and Scheaffer (1993) used the term Accessory maxillary sinus ostia for all the extra openings other than the primary maxillary ostium irrespective of their location. Levine et al (1993) described that accessory ostia

result from rupture of fonatanelles due to the blockage in the primary maxillary ostium. Scheaffer (1920) reported the AMO to number between 1 -3. But this is uncommon and hence there is not much in the literature regarding multiple openings. Stammberger & Kennedy (1995) came up with an incidence of 4-5 % in the general population as opposed to 25 % in those with chronic sinusitis.

Incidence of AMO ranges between 2 - 44 % , lower values being obtained from *in vivo* studies whereas higher ones from cadaver studies. The reason for this wide variation may be that the AMO that may be covered with a mucous film and in inaccessible locations are often missed out in *in vivo* studies. Whereas in cadaver studies, due to drying and fixing, the moist nasal mucosa can undergo shrinkage, also the fontanelle mucosa may be damaged during drying and investigations, giving an impression of higher incidence.

Schaeffer (1920) reported on an incidence of 43% in a cadaver study.

Myerson (1932), van Alyea (1936), Lang & Wurzburg (1991), Kumar. H *et al* (2001, Lady Hardinge medical college, New Delhi) in their studies on cadavers came up with an incidence of 31 %, 23 %, 28%, 30 %[5] respectively.

May et al (1990) found a 0 % incidence in cadavers and a 10 % incidence in endoscopic studies. Kennedy & Zinreicch in 1991 in an endoscopic study reported a value of 15 %. Wigand states that 25 % of specimens showed an accessory ostium[35].

INCIDENCE AND LOCATION OF ACCESSORY MAXILLARY OSTIA

S. No.	REFERENCE	LOCATION	INCIDENCE %	STUDY MATERIAL
1	Schaeffer (1920)	ANF or PNF	43	Cadavers
2	Myerson (1932)	Not Specified	31	Cadavers
3	Van Alyea (1936)	Not Specified	23	Cadavers
4	May et al (1990)	PNF	0	Cadavers
			10	Endoscopic
5	Kennedy &Zinreich (1991)	Not Specified	15	Endoscopic
6	Lang & Wurzburg (1991)	Not Specified	28	Cadavers
7	Stammberger & Kennedy (1995)	ANF or PNF	5	General population
			25	Diseased
8	Kumar et al (2001)	ANF/PNF/HS	31	cadaver

An experimental animal study was conducted by Genc et al in New Zealand type rabbits where they induced rhinogenic sinusitis using Streptococcus pneumonia in the right nasal cavities. After sacrificing the rabbits on the 21st day , their lateral nasal walls were examined. An accessory ostium was observed in 40% in the fontanelle like regions. The opposite nasal cavity showed no pus or accessory ostium. Histopathological examination of the mucosa showed - eosinophils, lymphocytes, plasma cells, ciliary loss, epithelial degeneration, areas of ulceration and lymphoid follicle hyperplasia.

RELEVANT ANATOMY

EMBRYOLOGY OF THE LATERAL NASAL WALL[2]

Paranasal sinuses develop from lateral nasal wall ridges called the Ethmoturbinals. In the 8th week of intrauterine life, 5-6 ridges are formed which undergo regression and fusion after which 3-4 persist. The 1stethmoturbinal regresses, with its ascending part forming the Aggernasi and the descending part forming the Uncinate. The second and third ethmoturbinals give rise to Middle and Superior turbinates, whereas the fourth and fifth fuse to form the supreme turbinate.

The Maxilloturbinal ridge arises inferior to these structures and gives rise to the Inferior turbinate. Primary furrows between the ethmoturbinals form nasal meati and recesses. The first furrow lies between the first and second ethmoturbinals, the descending part of which forms the Ethmoidal infundibulum, Middle meatus and Hiatus semilunaris. Its ascending part forms the Frontal recess. The second and third primary furrows form the superior and supreme meati respectively. Secondary lateral nasal wall Evagination gives rise to the Bulla ethmoidalis, and the Secondary lateral wall Invagination gives rise to the Supra and Retrobullar recesses.

Primordial maxillary sinus develops as a shallow groove from the inferior aspect of the Ethmoidal Infundibulum into the mass of the maxilla.[2,3] The evolution of maxillary sinus depicted in the following figure shows why the maxillary ostium is found at the floor of the ethmoidal infundibulum and why the drainage and ventilation of the maxillary sinus pass through it. Maxillary ostium lies on the medial wall of the ethmoidal infundibulum at the transition of its middle to posterior third.

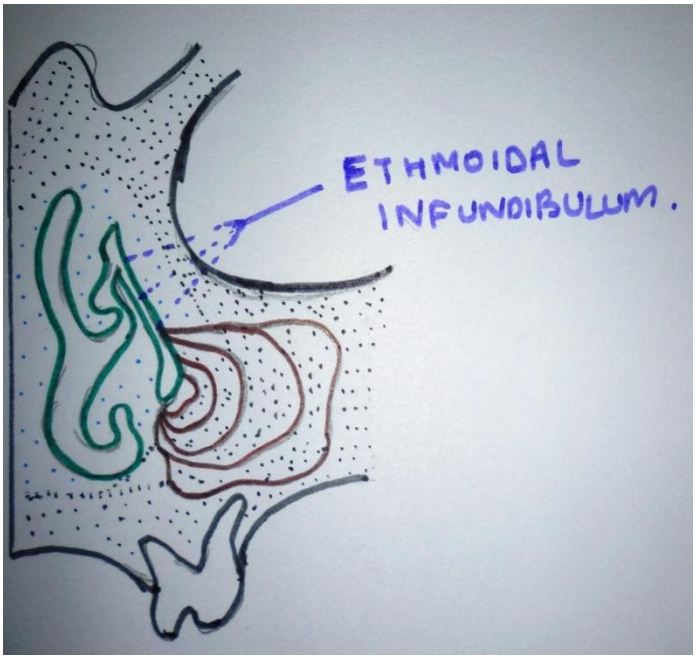


Figure 1:Development of maxillary sinus from ethmoidal infundibulum.

Maxillary sinus is the first sinus to appear (7 – 10 weeks).[3] It is around 7 x 4 x 4 mm^3 at birth. It grows at a rate of 2 mm vertically and 3 mm anteroposteriorly.[3] Its growth slows down around the age of 7 with a second growth phase thereafter.[3] At 12 years , pneumatisation reaches just under the lateral orbital wall at the junction of the zygomatic process. Inferiorly it reaches up to the level of the nasal floor , and , after 2^{nd} dentition , till below the nasal floor. [3] Its final size ~15 ml is attained at the age of 17 years.[3] Relative enlargement of the sinus occurs in old age due to resorption of the alveolus.

ANATOMY OF LATERAL WALL[2]

The complex ethmoidal labyrinth can be reduced into a series of lamellae which corresponds to the following - 1st- uncinate, 2nd – ethmoidal bulla, 3rd – basal or ground lamella and 4th – lamella of superior turbinate.

Agger Nasi

It is seen as a prominence anterior to the attachment of the middle turbinate to the lateral nasal wall. This region may be pneumatised by an anterior ethmoidal cell giving rise to the Aggernasi cell. It takes origin from the superior aspect of the infundibulum or the frontal recess region.

Ethmoidal Bulla

It is the most constant and largest of all anterior ethmoidal cells, located in the middle meatus , posterior to the uncinate anterior to the basal lamella. It is based on the lamina papyracea (LP). In 8% of subjects , it is unpneumatised and appears as a bony projection from the LP , known as the Torus Lateralis.

Hiatus Semilunaris

Hiatus Semilunaris Inferior of Grunwald is a 2 dimensional sagittaly oriented crescent shaped gap between the posterior free margin of the uncinate process and the anterior free margin of the bulla ethmoidalis. It communicates with the ethmoidal infundibulum.

Hiatus Semilunaris Superior

It is cleft between the posterior wall of bulla and the basal lamella where the middle meatus communicates with the Lateral Sinus (Retrobullar and Suprabullar recess)

Ethmoidal Infundibulum

It is a 3 dimensional funnel shaped passage through which secretions from Anterior ethmoid, Maxillary and frontal sinus are transported and channeled into the middle meatus.

Frontal Recess

It is the most anterosuperior part of the anterior ethmoidal sinus that forms a connection with the frontal sinus. It is bounded laterally by Lamina Papyracea, medially by middle turbinate,

anteriorly by the posterosuperior wall of Agger nasi and posteriorly by the anterior wall of the ethmoidal bulla.

Osteomeatal Unit

It is a functional designation (Naumann) referring to all the middle meatal structures ,viz. – the uncinate , ethmoid infundibulum , anterior ethmoidal cells , ostia of anterior ethmoidal , maxillary and frontal sinuses.

Uncinate process

It is a sickle shaped sagittal oriented structure paralleling the ethmoidal bulla and with a free posterior border. It is attached anterosuperiorly to the ethmoidal crest of maxilla. Directly inferior to this it is attached to the posterior aspect of the lacrimal bone. Posteroinferiorly its attached to the ethmoidal process of inferior turbinate ,and posterosuperiorly to the lamina perpendicularis of palatine bone. Superiorly it is attached to lamina papyracea / skull base / vertical attachment of the middle turbinate.

Fontanelles[1,2,3,8,35]

Anterior and posterior to its attachment to the inferior turbinate, uncinate process has no bony attachments. In a disarticulated skull there is a large opening, the maxillary hiatus, on the medial wall of the maxillary bone which in life is filled by the maxillary process of inferior turbinate, uncinate process, bulla ethmoidalis, perpendicular plate of palatine bone and lacrimal bone. Nevertheless a portion of this maxillary hiatus is devoid of bony structures and in life is covered by dense connective tissue which is a continuation of the periosteum and the mucous membranes of the middle meatus and the maxillary sinus. *Zuckerkandl* called these structures the *Anterior and Posterior Fontanelles* (AF &PF) in relation to the uncinate process. This part of the lateral nasal wall is also known as its *Membranous Area.* PF is larger and more distinct than AF and AO are frequently encountered in the PF. They are considered to be formed as a consequence of infection. Their formation is often likened to that of perforations on the tympanic membrane[3]. Their natural incidence is difficult to confirm yet there are views that it could be congenital. Their incidence

have been found to be 4-5 % in the general adult population and 25 % in those with Chronic rhinisinusitis[3].

MUCOCILIARY CLEARANCE[2]

Paranasal sinuses are lined by pseudostratified ciliated columnar epithelium with goblet cells. The maxillary sinus has the highest density of goblet cells compared to other paranasal sinuses (9700/mm^2).[3] Seromucinous glands are relatively infrequent , concentrated around the ostium. The thickness of the sinus mucosa is 0.2 – 0.8 mm.

Nasal cilia are relatively short (5 μ) and are found at the rate of 200/cell. They are formed of 9 paired outer microtubules surrounding a single inner pair of microtubules. They have a beat frequency of 12 Hz [2] (7-16Hz) [3] at body temperature , which propels materials at the rate of 3 – 25 mm / minute by their metachronous movement. Maxillary sinus produces remarkable quantities of Nitric oxide. The production is increased in cases of allergic rhinitis and decreased in chronic rhinosinusitis and Kartagener's syndrome. In chronic sinusitis ultrastructural changes in the mucosa with secondary ciliary dysfunction is seen , with 23%

mucosal samples showing absent ciliary activity. Isotope methods show that there is a lesser drainage of tracer substances in sinuses with

a. Retention of fluid

b. Thick mucosa

Quantitative and qualitative changes in the secretion, including the delicate periciliary fluid layer is of greater importance for the impairment of mucociliary transport during sinus inflammation than the structural abnormality of the cilia or their retarded beat rate. Ciliary impairment in the presence of purulent secretions is due to-

a. High proteolytic enzyme activity

b. Low pH

c. Anaerobic mucosal metabolism

d. Impairment of mucosal oxygen supply (rare)

SECRETION AND TRANSPORTATION[1]

Principles

Drainage and ventilation are two important factors for the normal physiology of the paranasal sinuses and their mucus membranes. Drainage depends on secretion and transport mechanisms which in turn depend on -

- Amount of mucus
- Composition of mucus
- Effectiveness of ciliary beat
- Mucosal resorption
- Condition of ostia and ethmoidal clefts
- Free flow of inspired air
- Mucosal pulsations and movements of fontanelles (in case of inflammation)

The nasal mucosa is lined by a mucous blanket which is produced by the mucoserous nasal glands and intraepithelial goblet cells. Mucus is a complex non Newtonian fluid whose quality and

quantity are important and requires an intact blood supply and nervous system. It is composed of -

1. Water & ions
2. Glycoproteins (sialomucins , fucomucins , sulphomucins)
3. Enzymes (lysozymes, lactoferrin)
4. Circulatory proteins (Complement , α 2 macroglobulin , CRP)
5. Ig- IgA , IgE , IgG , IgM , IgD
6. Cells – surface epithelium , basophils , eosinophils , leucocytes.

The mucus film has two layers – the *sol* phase , which is the *inner serous* layer (water and ions) and the *gel* phase , which is the *outer* viscous layer (glycoproteins). Cilia beat in the inner sol phase and the outer gel phase also moves along over the sol phase like a "carpet". Dust particles adhere to the gel phase for being transported out of the paranasal sinuses. A pH of range of 7.5 – 7.6 is required for maintaining an equilibrium between the sol phase and gel phase. The production and composition of this mucus is dependent on –

- Humidity
- Pollution
- Airborne irritants

The mucosal glands are controlled mainly by the parasympathetic fibers. The nerve fibers from the Superior Salivatory Nucleus via the Greater Petrosal Nerve reach the Pterygopalatine ganglion, from where the postganglionic fibers supply the mucosal glands.

The sympathetic fibers arise from the Lateral Horn of the spinal cord. The postsynaptic fibres via the Carotid plexus form the DeepPetrosal Nerve which joins the Greater Petrosal Nerve to form the Vidian Nerve which ultimately supplies the nasal and sinus mucosa.

Substance P secreted from type C fibers via local reflexes also have great effect on mucosal glands. They are found to produce Hypersecretion , Vasodilation and extravasation of plasma.

On an average the maxillary sinus mucus layer is renewed every 20 – 30 minutes. The mucosal secretions form a homogenous layer all along the walls of the sinuses except at the ostia where

the viscous layer is found to be thicker as secretions from the whole sinus converge there.

When mucosal surfaces come close to each other leaving a recess in between, the gap is filled by the mucosal blanket by a the *Bridging phenomenon*, whereby the cohesive forces in the gel phase bridge the gap, while the sol phase fills the recess in between. Similarly the flow over a small mucosal defect also goes unhindered due to cohesive nature of the mucus carpet.

But when the mucus is too viscous, this defect can prove to be an obstacle with the secretion being retained at the site of the defect. Similarly at the region of crests a thick secretion will be retained for a while and finally drain away under the influence of gravity. A highly viscous mucus can block the primary maxillary ostium ,later fall down into the sinus only to be transported again towards the primary ostium. If the sinus ostium is oval or oblong , theciliary beat works on the mucus from two or three sides and the mucus passes through the corners of the ostium.

In cases where an Accessory ostium of up to 4mm is present , a normal secretion bypasses it, and a viscous secretion moves over

it without leaving the sinus through it. But in larger accessory ostia the part of the mucus passing through its middle gets transported out, whereas that moving around the corners get bypassed. This same phenomenon can be visualized in inferior meatal antrstomies. Accessory ostia also are sites of what is called *'recirculation'*. ie. Mucus from the ethmoidal infundibulum moves into the maxillary sinus via the accessory ostium and then out through the natural ostium only to return back through the accessory ostium.

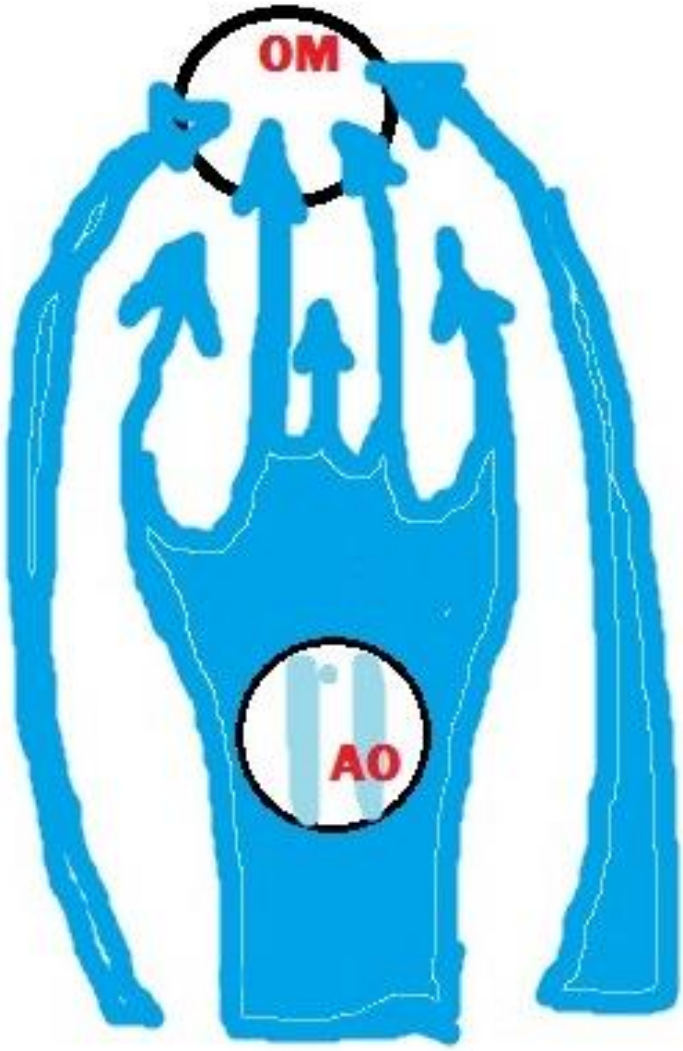


Figure 2Mucous Blanket moving over and across the Accessory Ostium towards the Natural Ostium

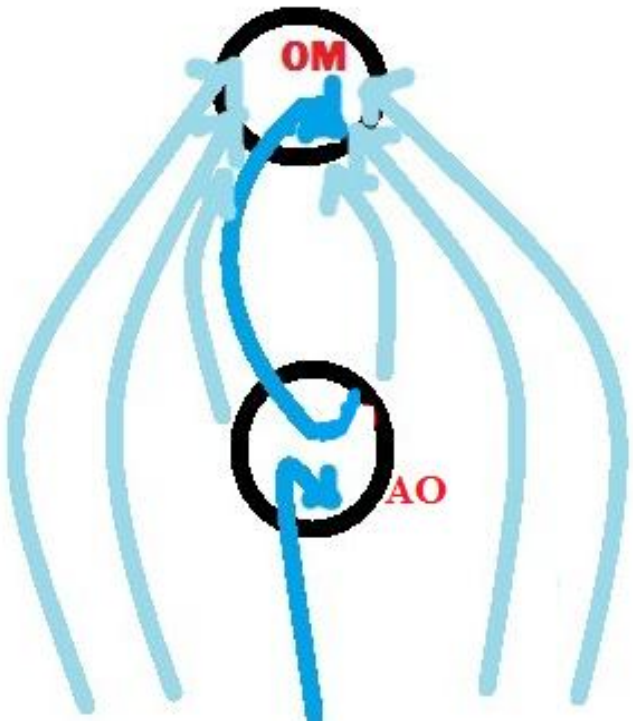


Figure 3Blue arrow shows circular transport of mucous.

SECRETION TRANSORT PATHOLOGY

Hypersecretion causes the mucus to flow into the deepest part of a sinus due to gravity. In a normally composed mucus , the gel layer persists on the surface. Active cilia in the non drowned part of the mucosa may pull away the mucus carpet on the surface because of cohesion , provided the cilia beat normally and the

direction of transport in the corresponding areas are not opposing each other.

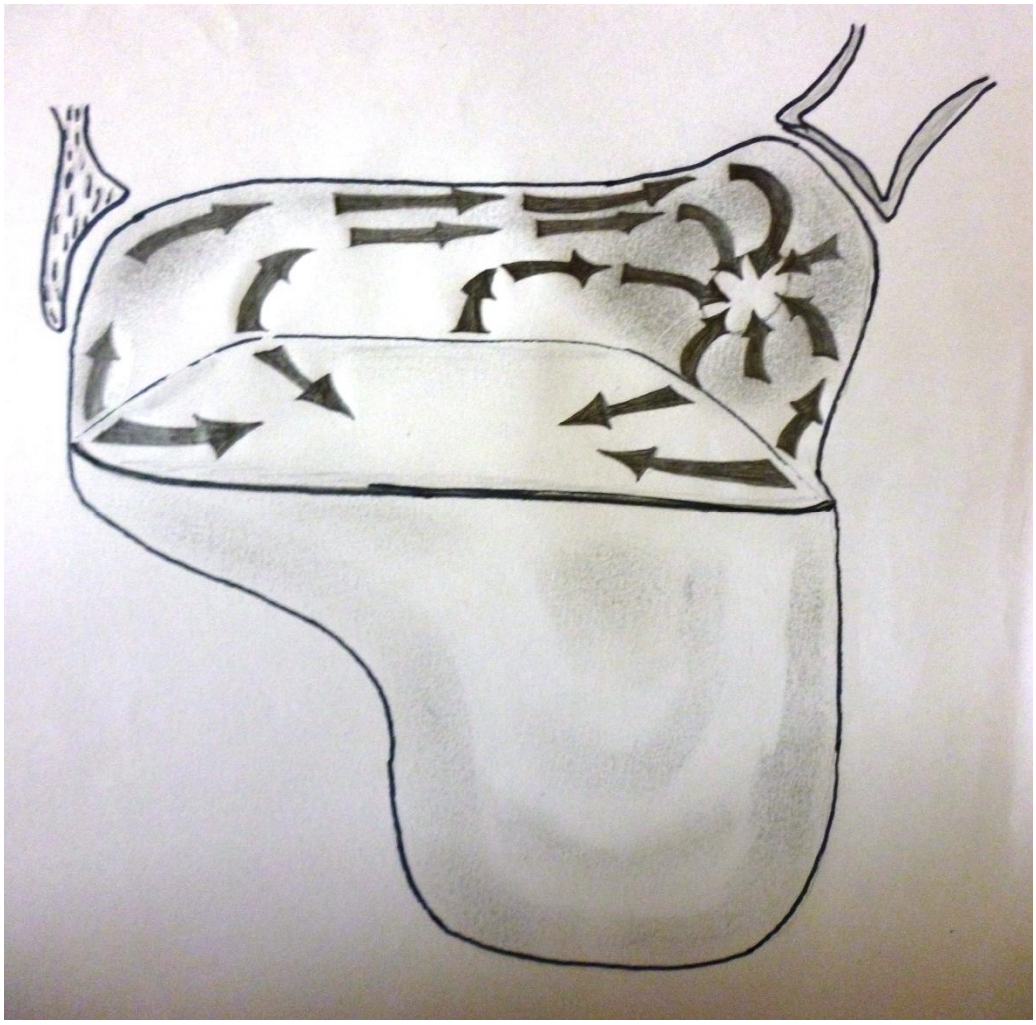


Figure 4 A half filled maxillary sinus. The cohesional forces of the gel phase are holding back the transportation of the mucus. The still intact cilia are not powerful enough to 'tear off' the gel layer from the surface

In case of decreased secretions or reduced humidity at the surface , sol phase becomes rather thin. The mucus becomes very viscous and the gel phase comes in direct contact with the cilia , thereby impeding their action resulting in a worm like movement of the mucus layer.

During inflammation of the sinuses , the mucosa gets inflamed rapidly and even pulsate. Also increased movements in the region of the membranous portions of the fontanelles may occur. Such mucosal movements may also assist the transport of secretions out of the maxillary sinus.

There is another phenomenon known as "*secretion expressways"* which is found in both cases of abnormal secretions as well as apparently normal sinuses. This means that the transport of secretion is not uniform throughout a sinus. From time to time the mucosa of one region transports secretion faster than the neighboring areas. In some time the slower areas catch up speed and the faster region slows down.

The normal ciliary beat frequency ranges between 8 – 20 beats per second. Optimal mucociliary clearance system requires –

- Normal ventilation
- Humidification
- Normal Metabolism
- Osmotic pressure
- Optimal pH – 7-8
- Optimal temperature – around 33^0 C
- protection from noxious stimuli

TRANSPORT OF SECRETION IN THE MAXILLARY SINUS

Hilding Sr. and later Messerklinger made the discovery that secretions from sinuses follow a genetically predetermined route to reach their respective ostia. In the maxillary sinus the secretions are transported in a *stellate* pattern from the floor. The secretions pass along the anterior , medial posterior , lateral walls and the roof of the sinus and finally converge at its natural ostium. From there it moves to the floor of the posterior third of the ethmoidal infundibulum where the natural ostium usually opens. In the ethmoidal infundibulum the secretions from the frontal and anterior

ethmoidal sinuses usually join. The ethmoidal infundibulum via the Hiatus

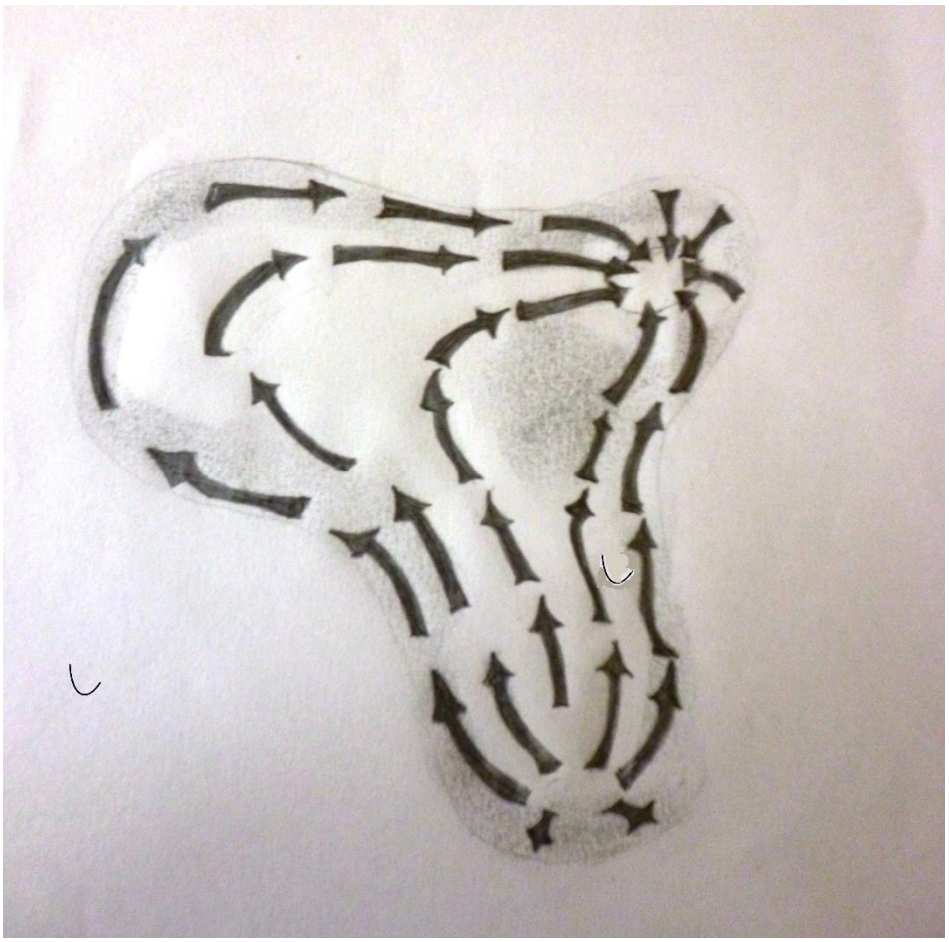


Figure 5Mucous Transportation Pathways inside Maxillary Sinus

semilunaris drains into the middle meatus. From here the secretion passes over the posterior free border of the uncinate process and along the medial face of the inferior turbinate and then into the nasopharynx posteriorly where they pass anterior and inferior to the Eustachian tube orifice. Up to the border of the

ciliated and squamous epithelium active transport continues after which secretions move downwards by gravity aided by the swallowing mechanism.

Secretions from the maxillary sinus always tend to pass through the natural ostium even in the presence of a single or multiple accessory ostia or a surgically created inferior meatal antrostomy.

MECHANISM OF MAXILLARY SINUSITIS

During the transport of secretions through the ethmoidal infundibulum, which is a prechamber through which the maxillary sinus communicates with the middle meatus, if its opposing mucosal surfaces come into intense contact and firmly pressed against each other pathologically , the cilia in the region get immobilized and mucus transport is hampered. Obstruction in such a key area acts like a 'bottle neck' and affects the ventilation and drainage of the whole maxillary sinus resulting in retained secretions. When this area of obstruction expands or an infection ensues the retained secretions acts as a culture medium for bacteria or virus , thus perpetuating the vicious cycle.

Also poor ventilation decreases the pH of the sinus , further slowing down its ciliary movement with the resultant formation of viscous mucous. Such viscous mucous may remain in the sinus for a considerable length of time due to the blockade in the prechamber. This again is a favorable condition for microbial growth, toxins of which may cause further impairment of the mucosal function. This is an instance where an otherwise normal sinus ends up diseased due to a pathology that lies in the outflow tract.

Inhaled pathogens deposited at the entrance of the middle meatus adhere to the mucus. Due to the confluence of the mucus pathways of the ethmoidal infundibulum and the frontal recess , the microbes get transported into either of the sinuses. In the presence of a suboptimal self healing capacity of the sinus mucosa or inadequate antibiotic therapy an Acute or a Chronic recurring sinusitis results.

Maxillary sinuscopic studies have revealed that viscous mucus sometimes enters the sinus through accessory ostiapresent in either of the fontanelles. This mucus is transported along the natural pathways towards the natural ostium where it exits the sinus. This

mucus may again re enter the sinus via the accessory ostium and this may continue endlessly. As long as the natural ostium is patent this is not of much significance. But if it is blocked or in the presence of a nasal infection this plays an important role in the transport of pathogens into the maxillary sinus from the nose. Due to ostial blockade infected secretions are unable to leave the sinus resulting in maxillary sinusitis.

Thus the cause of infections of the large sinuses like maxillary sinus is mostly rhinogenic. It usually spreads from the nose through the ethmoid clefts and prechambers into the maxillary and frontal sinuses. An exception to this is dentogenic sinusitis (<2%). Other such exceptions are foreign bodies in the maxillary sinus (aberrant root filling material) , blood in the sinus following trauma, cholesterol or mucus retention cysts (depending on their location).

OSTIAL PATENCY AND GAS STUDIES[2]

A normal average sinus ostial diameter is around 2.5 mm (3 – 6 mm) and 6mm in length. When ostial patency is impaired (mucosal swelling, mucus entrapment, polyps etc) there is an

aberration of pO_2 and pCO_2 which results in mucosal exudation and development of local pathology. Mucosal exudation is preceded by extravasation and interstitial spreading of plasma derived liquids and proteins leaving the lamina propria with a pool of inflammatory mediators.

Clinical initiation of sinusitis is a 3 step process -

- Ostial obstruction
- Pathogenic bacteria
- Impaired local defense.

In vivo studies and measurements of sinus ventilation are rare owing to their small size and inaccessibility of the cavities. Assumption of a uniform pressure throughout the nose led Proetz (1932) to state that the presence of an accessory ostium could not increase the exchange rate of sinus air. In a study conducted by Hood *et al*, Departments of Bioengineering, Aeronautics and Otolaryngology, St. Mary's Hospital , Imperial college London[4], they created numerical models of human maxillary sinuses to understand the sinus gas exchange better. They found that, in the presence of a small ostium and a large concentration difference between the nose

and sinus, diffusion is the dominant transport mechanism. Whereas for larger ostia and smaller concentration differences it is convection. Contrary to Proetz's findings , they have proved that the presence of one or more accessory ostia increases the sinus ventilation rates by several folds. In their study model, a standard *double ostium* geometry (*ostium 1 &ostium 2 with 3mm diameter & 6 mm length each)* has a pressure difference of ~0.1 Pa between the ostia.[4] This pressure difference produces an inflow to the sinus through the upstream ostium and an outflow from the sinus through the downstream ostium of 7.2 x $10^{-7}m^3/s$.[4] The time to replace 90 % of sinus air (T_{90}) ~31.9 seconds. In the various other double ostium models , which varied in the ostial diameter and length , this T_{90} varied from 8.9 seconds to 31.9 seconds.[4]

LIMITATIONS OF SINUS VENTILLATION[4]

The natural ventilation rate in a single ostium sinus is extremely slow. This slow ventilation is protective as it -

- helps prevent drying of mucosal secretions
- helps maintain a near sterile environment with high NO concentration and minimal pathogen access.

Whereas the transport rates are higher in a double ostium sinus with ostia exposed to even very slightly different pressure changes compared to a single ostium sinus. When a sinus with a single ostium can be considered as a reservoir of fluid attached to the nose, only a sinus with multiple ostia can have a net flow through it as it offers an alternative flow path in parallel with the nose. Thus their study contradicts Proetz's conclusion that an accessory ostium cannot increase the ventilation of a sinus.

ADVERSE EFFECTS OF INCREASED VENTILATION

1. Decreased NO concentration - (when convective transport exceeds production) leading to impaired mucociliary function resulting in increases pathogen entry into the sinus

2. Mucosal drying - (as density of goblet cells in sinus is less than nasal cavity). Risk of mucosal drying is more when the upstream ostium is closer to the nostril.

3. Circular mucociliary transport - by this pathogen from other parts of the nasal cavity gains access into the sinus.

5. MATERIALS AND METHODS

83 Patients (Male and female in the age group of 15-45 years) with Chronic Maxillary Sinusitis who attended the ENT Out Patient Department and 83 subjects , who attended the ENT Out Patient Department for reasons other than Chronic Sinusitis and normal volunteers, during the period January 2013 to July 2014 , who satisfied the inclusion criteria were enrolled for the study after getting an informed written consent.

Study design : Cross Sectional study

Study place : Department of ENT, Government Stanley Medical College and Hospital.

Study and Follow-up period : January 2013 to July 2014.

Sample size: exposed subjects – 83
unexposed subjects – 83
Total sample size – 166

METHODS OFSTUDY

- HISTORY
- CLINICAL EXAMINATION
- DIAGNOSTIC NASAL ENDOSCOPY

DIAGNOSTIC CRITERIA FOR CHRONIC RHINOSINUSITIS(1997 RHINOSINUSITIS TASK FORCE)[9]

(>12 weeks)

Major Factors

1.Facial pain/pressure

2.Nasalobstruction

3.Nasaldischarge/discolored postnasal drip

4.Hyposmia/anosmia

5.Purulenceon examination

Minor Factors

1.Headache

2.Fever (all nonacute)

3.Halitosis

4. Dental pain

5. Fatigue

6. Cough

7. Ear pain/pressure/fullness

DIAGNOSIS[9]

Presence of either;

- 2 major factors, or,
- 1 major and2 minor factors.

Facial pain/pressure alone does not constitute a suggestive history for diagnosis in the absence of another major symptom or sign.

Since in our present study we had normal subjects as well , we did not use the CT findings as a criteria in order to avoid unnecessary radiation exposure. Hence in our study we only adhered to the Task Force Criteria and Nasal Endoscopic findings for diagnosing Chronic Sinusitis.

EQUIPMENT USED:

1. 4mm – wide angled zero degree, 30^{o} Karl Storz endoscopes.
2. Stryker HD camera (24 mm)
3. High definition LED monitor

Patient in supine position with head turned to right side.

Figure 61.HD camera , 2. 30^0 Karl Storz Endoscope , 3. 0^0 Karl Storz endoscope , 4. Tilley's nasal dressing forceps , 5. Thudicum speculum , 6. Metal suction tip , 7. Kidney tray , 8. Nasal packs dipped in decongestant solution , 9. Defog solution – Savlon

Figure 7Diagnostic Nasal Endoscopy being done in our Department

PROCEDURE[10,11]

- Topical anesthetic, about 7 ml of 4% xylocaine was mixed with 10 drops of xylometazoline. Cotton pledgets dipped in the solution, squeezed dry and used to pack the nasal cavity. Pledgets were packed in the inferior, middle and superior meati and left in place for full 5 minutes. Diagnostic endoscopy is performed using a 4mm30 degree Hopkin nasal endoscope.

- First pass :

  The endoscope was introduced along the floor of the nasal cavity. First the inferior meatus came into view. In cases where the inferior turbinate was lateralized , the same was medialised by applying more topical anaesthetic. Endoscope was advanced posteriorly to identify adenoid tissue. Eustachean tube identified. The entire nasopharynx was visualized by rotating the 30^0 endoscope.

- Second pass:

  Next, the scope was gently withdrawn out and slid medial to the middle turbinate, between the middle turbinate and nasal septum.

The scope is gently slipped medial to the middle turbinate to view the sphenoethmoidal recess.

- Third pass:

The shape and size of the middle turbinate as well as its relationship to the lateral nasal wall and septum was evaluated. The middle turbinate is gently medialised and the attachment of the uncinate process is carefully noted. Any discharge in this area also recorded. *If accessory ostium is present it comes into view now. Accessory ostium is present more posteriorly. Normal ostium is actually not visible during diagnostic nasal endoscopy. Accessory ostium is spherical in shape and oriented anteroposteriorly, while the natural ostium of maxillary sinus is oval in shape and oriented transversely.*

PMO	**AMO**
Ovoid in shape	Circular
Anterior and superior to AMO	Posrerior and inferior to PMO
Always present	Not present in all cases
Difficult to see clinically	Easily seen if present

CROSS SECTIONAL STUDY

A cross-sectional study examines the relationship between disease (and other variables of interest as they exist in a defined population at a single point in time or over a short period of time. Cross-sectional studies can be thought of as providing a snapshot of the frequency of a disease or other health related characteristics (e.g. exposure variables) in a population at a given point in time.

Cross-sectional studies are used to assess the burden of disease or health needs of a population and are particularly useful in informing the planning and allocation of health resources.

SAMPLE SIZE CALCULATION

From the review of literature on earlier studies on the presence of accessory ostium, we chose Kennedy and Stammberger's endoscopic study which gave a prevalence of 5% in the general population[5]. In a study conducted in the Rajiv Gandhi University of Health Sciences , Karnataka the prevalence of accessory ostia in chronic sinusitis was found to be 22 %[46] In order to get an Indian standard the value of 22% was used for calculation.

Sample size was calculated using OpenEpi Version 3 , open source calculator.

Sample Size:X-Sectional, Cohort, & Randomized Clinical Trials

Two-sided significance level(1-alpha):	95
Power(1-beta, % chance of detecting):	90
Ratio of sample size, Unexposed/Exposed:	1
Percent of Unexposed with Outcome:	5
Percent of Exposed with Outcome:	22
Odds Ratio:	5.4
Risk/Prevalence Ratio:	4.4
Risk/Prevalence difference:	17

	Kelsey	**Fleiss**	**Fleiss with CC**
Sample Size – Exposed	85	83	95
Sample Size-Nonexposed	85	83	95
Total sample size:	170	**166**	190

$$\text{Prevalence} = \frac{\text{number of cases in a defined population at a given period of time}}{\text{number of persons in the given period of time}}$$

Using the formula the prevalence of accessory ostium in chronic sinusitis (P_c)and the prevalence of accessory ostium in those without chronic sinusitis (P_n) was calculated.

P_c = 30 %

P_n= 10 %

In order to find the significance of the values , a Chi Square test was done on the sample.

The data captured in the sample size was arranged in the following table to arrive the X^2

	Data type 1	Data type 2	Totals
Category 1	A	b	a + b
Category 2	C	d	c + d
Total	a + c	b + d	a + b + c + d = N

The formula for Chi Square Distribution is

$$X^2 = \frac{(ad-bc)^2 (a+b+c+d)}{(a+b)(c+d)(b+d)(a+d)}$$

	AO +	AO -	Totals
Exposed (CRS +)	25	58	83
Not Exposed(CRS -)	8	75	83
Total	33	133	166

Chi Square distribution table.

probability level (alpha)

Df	0.5	0.10	0.05	0.02	0.01	0.001
1	0.455	2.706	3.841	5.412	6.635	10.827
2	1.386	4.605	5.991	7.824	9.210	13.815
3	2.366	6.251	7.815	9.837	11.345	16.268
4	3.357	7.779	9.488	11.668	13.277	18.465
5	4.351	9.236	11.070	13.388	15.086	20.517

Applying the formula above we get:

Chi square = $166[(25)(75) - (8)(58)]^2 / (33)(133)(83)(83) = 10.93058$

When a comparison is made between one sample and another, a simple rule is that the degrees of freedom equal (number of columns minus one) x (number of rows minus one) not counting the totals for rows or columns.

For our data this gives (2-1) x (2-1) = 1.

We now have our chi square statistic ($x^2 = 10.93058$), our predetermined alpha level of significance (0.05), and our degrees of freedom (df = 1). Entering the Chi square distribution table with 1 degree of freedom and reading along the row we find our value of x^2 ($x^2 =$

10.93058) lies near 10.827. The corresponding probability is less than 0.001. Since a p-value of 0.001 is lesser than the conventionally accepted significance level of 0.05 (i.e. $p < 0.05$) we reject the null hypothesis.

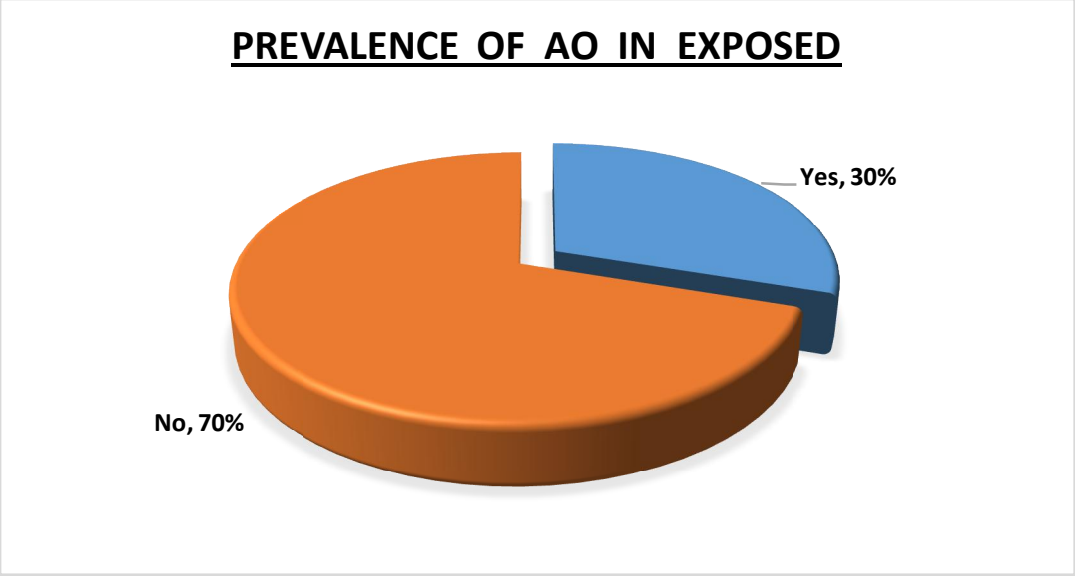
In other words, there is a statistically significant difference in the proportion of AO in patients with Chronic Sinusitis and in those without.

When $p < 0.05$, we generally refer to this as a significant difference.

6. RESULTS

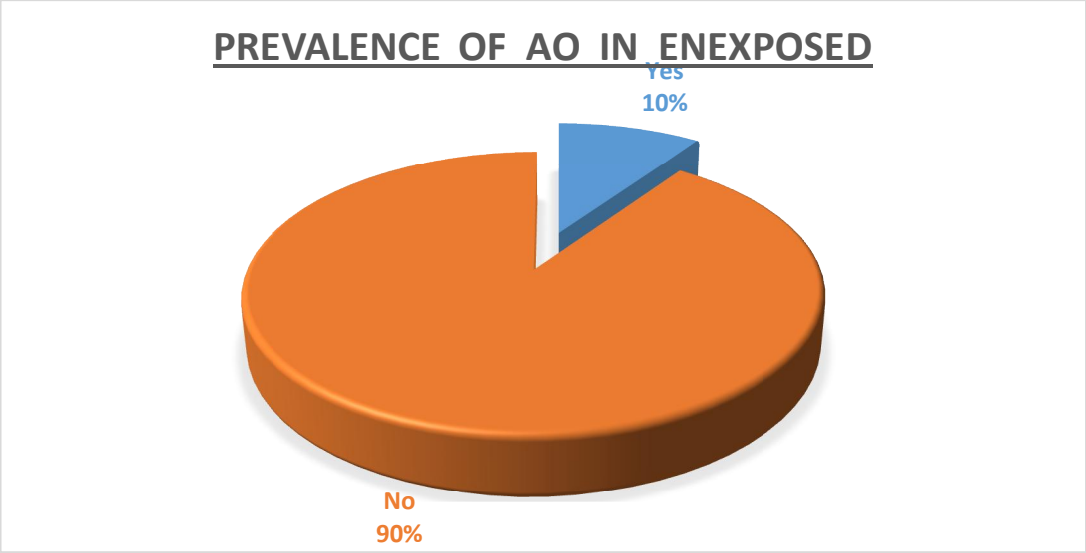
Prevalence Of AO (Accessory ostium) In The Exposed Population

AO	No. in exposed population (CRS)
YES	**25**
NO	58
TOTAL	83

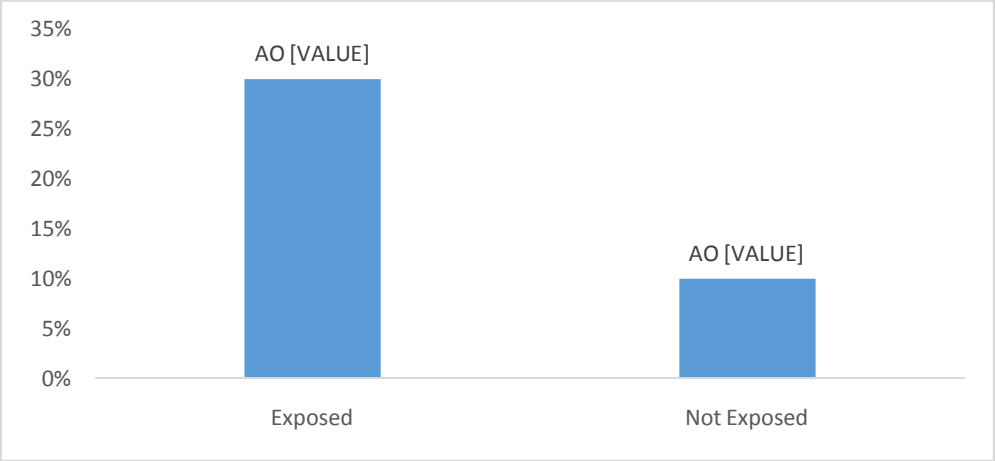


Prevalence Of AO In The Unexposed Population

AO	No. in unexposed population
YES	**8**
NO	75
TOTAL	83



Bar Chart Showing In Difference In Prevalence In Chronic Sinusitis &Normal Population



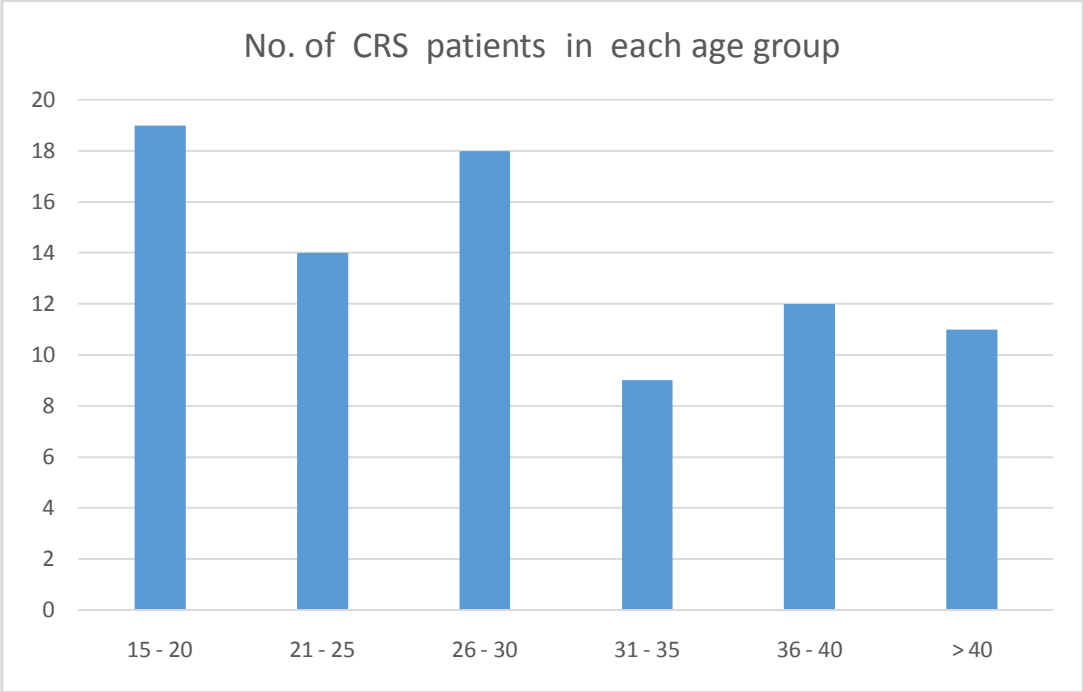
Among the 83 CRS patients enrolled for study , 25 (30 %) had an Accessory ostium and in the 83 unexposed persons it was 8 (10 %)

AGE DISTRIBUTION OF AO IN CHRONIC SINUSITIS (CRS)

Subjects in the age group 15 – 45 years were chosen for the study. The distribution of CRS in the various age groups , as obtained from our recent study has been shown in the table and chart are given below.

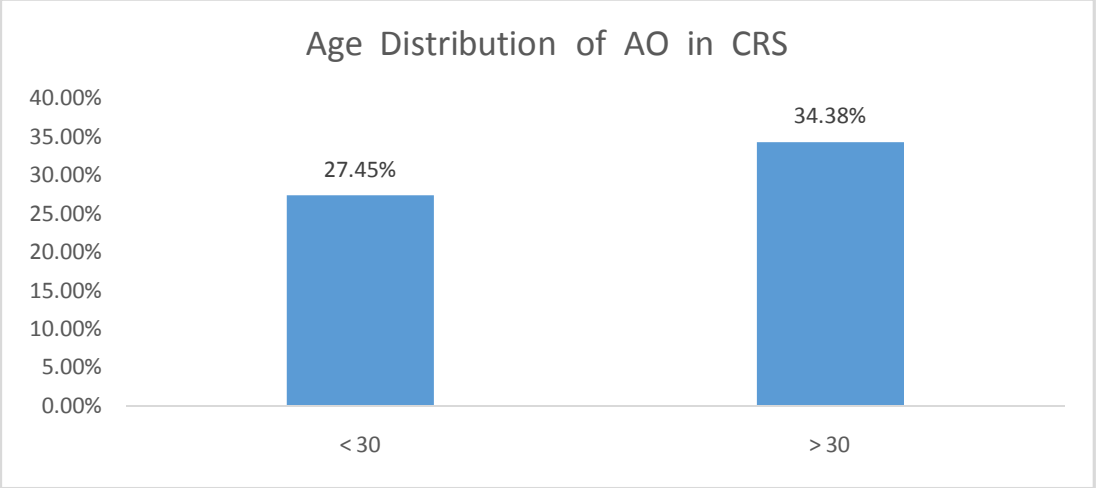
Age distribution of CRS

Age group	Frequency of CRS
15 - 20	19
21 - 25	14
26 - 30	18
31 - 35	9
36 - 40	12
40 - 45	11



Age Distribution of AO in CRS

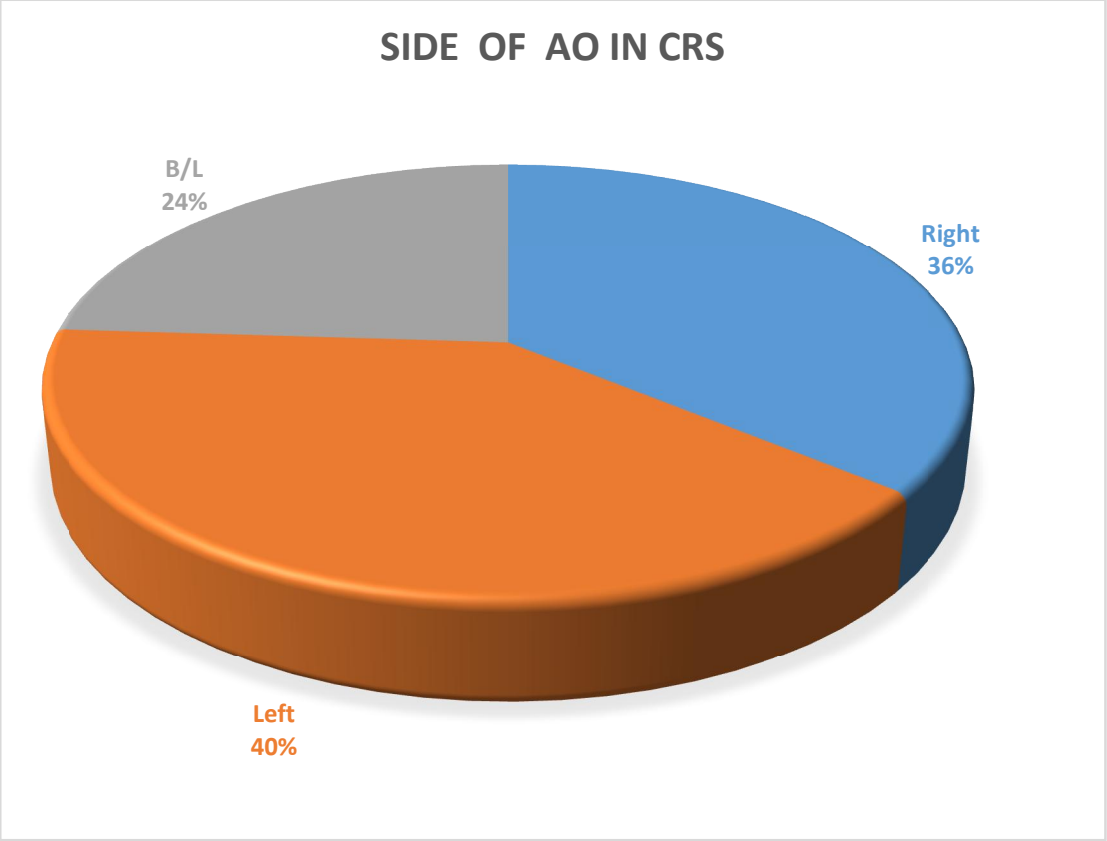
	CRS	AO	% of AO
Age < 30	51	14	27.45
Age > 30	32	11	34.38



SIDE DISTRIBUTION OF AO IN CHRONIC SINUSITIS

The following are the tables and charts showing the side on which the AO was present - right / left / bilateral in the group with CRS.

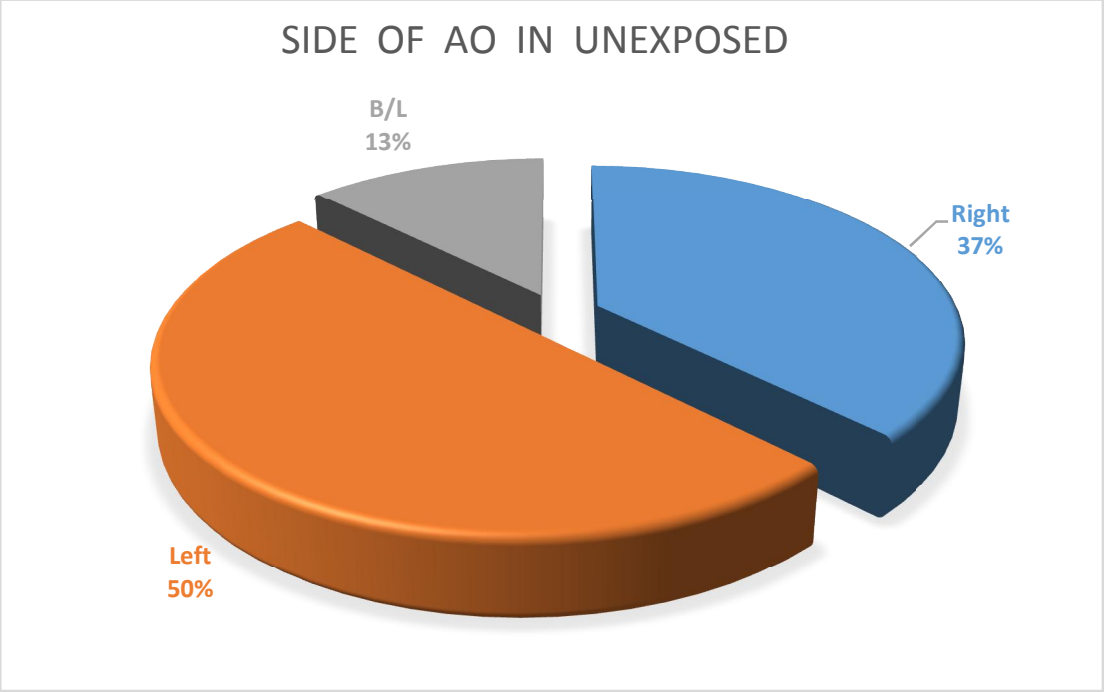
SIDE	FREQUENCY
Right	9
Left	10
B/L	6



SIDE DISTRIBUTION OF AO IN THE UNEXPOSED

The following are the table and chart showing the side on which the AO was present - right / left / bilateral in the unexposed group.

SIDE	FREQUENCY
Right	3
Left	4
B/L	1

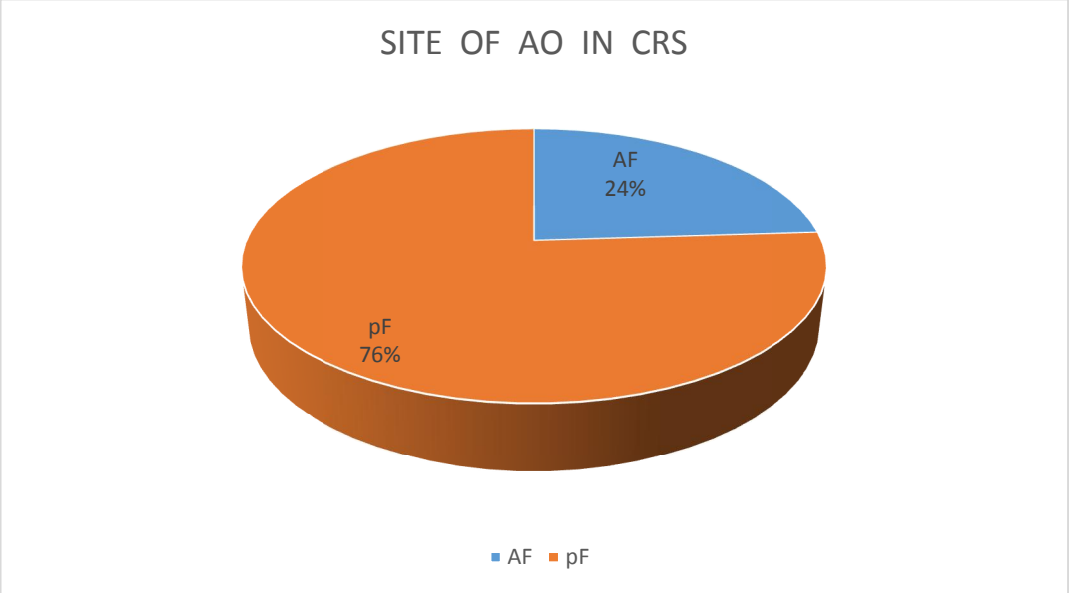


DISTRIBUTION OF AO ACCORDING TO ITS SITE ON THE LATERAL WALL – ANF / PNF

AO is more commonly found on the posterior nasal fontanelle (PNF) than the anterior nasal fontanelle (ANF). Following is the frequency of the sites of occurrence in the two groups.

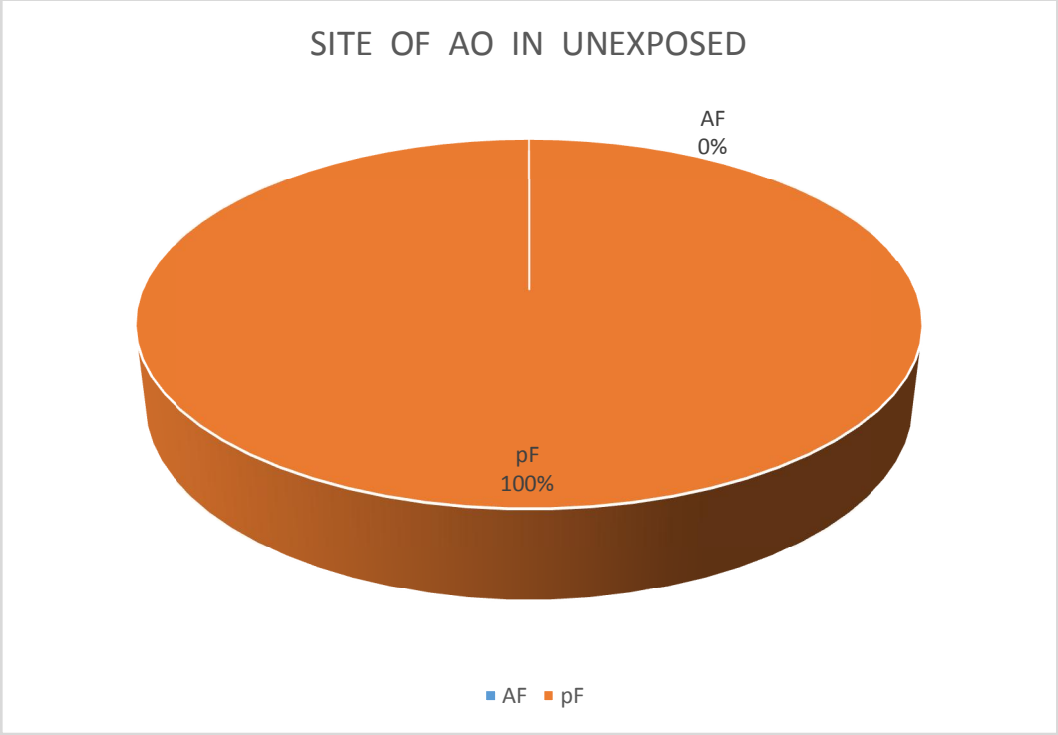
IN CHRONIC SINUSITIS

SITE	FREQUENCY
ANF	6
PNF	19



IN THE UNEXPOSED

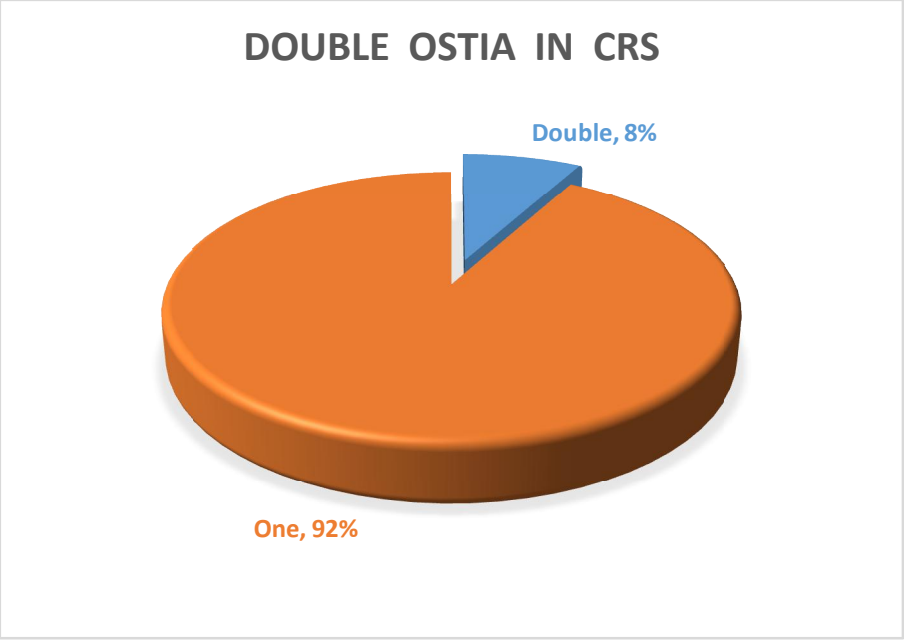
SITE	FREQUENCY
ANF	0
PNF	8



DISTRIBUTION OF DOUBLE OSTIA IN CHRONIC SINUSITIS

The occurrence of AO is usually single and occasionally multiple. In our study we found only 2 such cases and that too only in those with CRS. Double ostia were not present in the unexposed group in the present study.

NO. OF OSTIA	FREQUENCY
DOUBLE	2
SINGLE	23

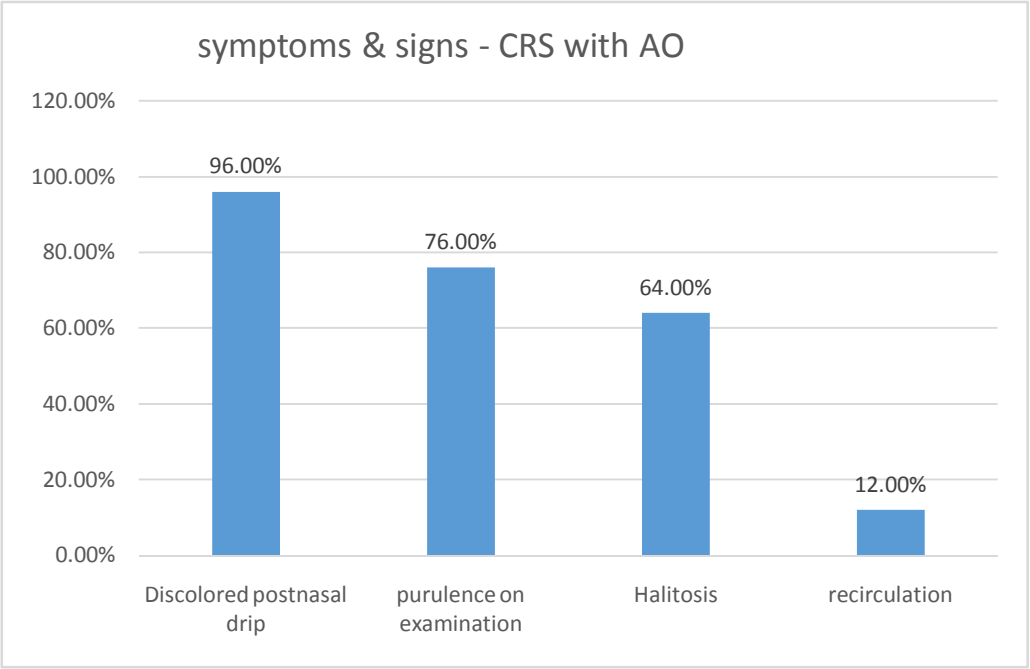


Distribution of signs & symptoms among CRS patients with and without AO

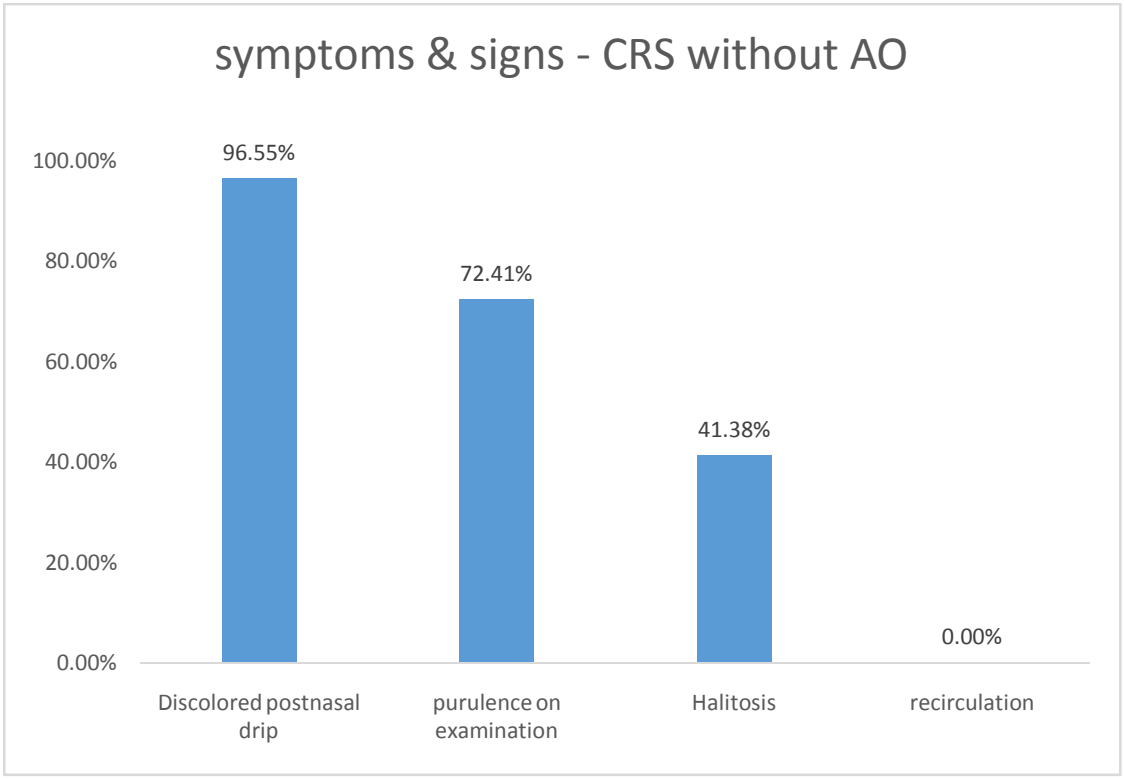
The following tables show the variation in percentage of symptoms and signs like *discolored postnasal drip , halitosis , purulence on examination.*

Also *Recirculation phenomenon* in the accessory ostium was found only in 2 patients of CRS.

	Frequency	CRS with AO	Percentage
Discolored postnasal drip	24	25	96.00%
Halitosis	16	25	64.00%
Purulence on examination	19	25	76.00%
Recirculation	3	25	12.00%



	Frequency	CRS without AO	Percentage
Discolored postnasal drip	56	58	96.55%
Halitosis	24	58	41.38%
Purulence on examination	42	58	72.41%
Recirculation	0	58	0.00%


symptoms & signs - CRS without AO
100.00%
80.00%
60.00%
40.00%
20.00%
0.00%
96.55%
72.41%
41.38%
0.00%
Discolored postnasal drip
purulence on examination
Halitosis
recirculation

7. DISCUSSION

INTERPRETATION AND ANALYSIS OF DATA

PREVALENCE OF ACCESSORY OSTIUM

Analysis of our present study shows that 25 out of 83 ie. **30 %** CRS patients had an AO and that 8 out of 83 ie. **10 % ,** subjects without CRS had an AO. All of them were ***round*** in shape. As mentioned earlier prevalence of AO shows a wide range between 2 – 44 % , with cadaveric studies showing a higher incidence than that on live subjects. The various studies and results have been tabulated in the section 'Review of Literature'.

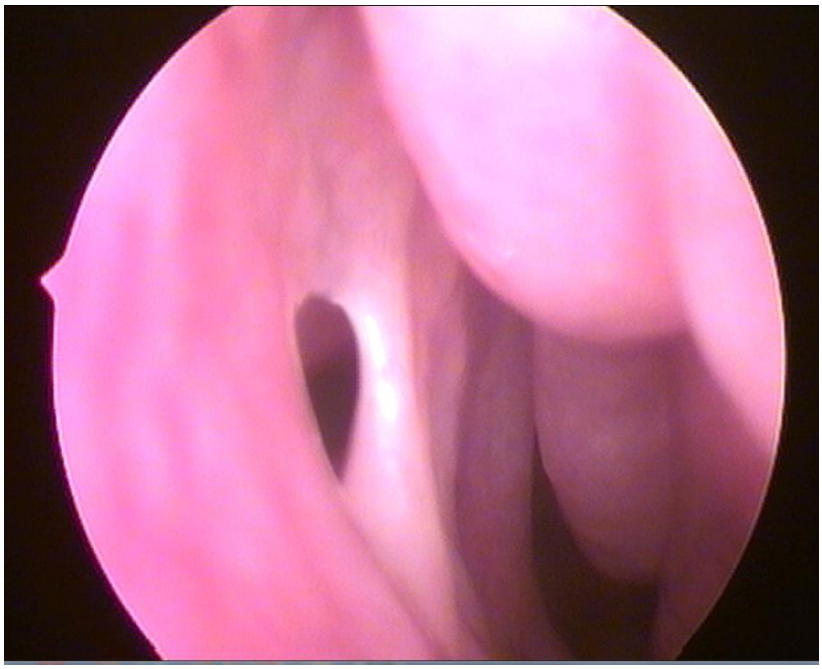


Figure 8a Right sided AO in a patient with CRS

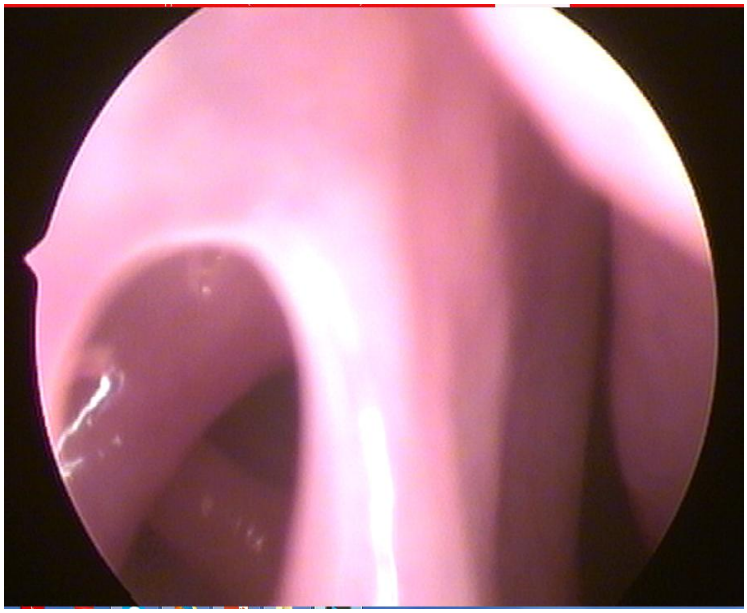


Figure 9 A closer look at the same AO showing the interior of maxillary sinus

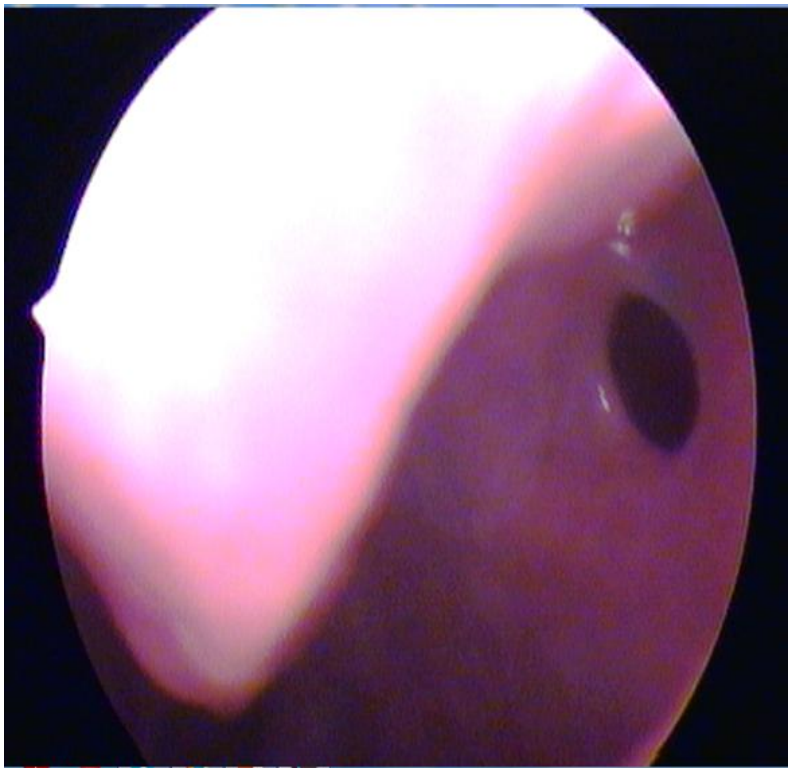


Figure 10Showing 23 year old male without CRS with left PNF AO

Here we are quoting a few more studies that were not discussed in the previous section.

In a prospective cohort study by Jog and Mc Garry, on Rhinology clinic patients and General ENT clinical controls, they reported that 7 % of Rhinology patients and 2% of the controls had AO. Of the rhinology patients with rhinitis and sinusitis , 8 % showed AO.[20]

AlperSindel et al [21] Manju et al [19] Kolvekar et al[22] Manjula Patil et al[23]in their cadaver studies have reported the prevalence as 13.8% , 18.5 % , 22.5% and 26% respectively.

AGE

Subjects chosen for the study belonged to the age group between 15 and 45 years. In the present study the frequency of CRS in each group was found to be the following –

51patients with CRS were below the age of 30, and 32 were above the age of 30. Of the 51 patients below 30 years, 14 had an

accessory ostium, ie. 27.45 %. Of the 32 patients above 30 years, 11 had accessory ostia, ie. 34.38 %.

In the unexposed group, of the 35 individuals below 30 years 4 (11.42 %) had AO, and out of 48 above 30 years, 4 (8.33 %) had AO.

Though literature says that AO can develop with advancing age, such an association was not obtained in our study[24].

GENDER

According to the review of literature gender had no effect on the development of AO. Hence no conscious effort was taken to equalize the number of males and females while enrollment.

SIDE

In our study, of the CRS patients with AO, 10 (**40** %) on the **Left,** 9 (**36** %) were on the **Right** and 6 (**24** %) were present **Bilaterally**. And in the unexposed group, 4 (**50 %**) on the **left,** 3 (**37%**) on the **right**, and 1 (**13 %**) were found **bilaterally**.

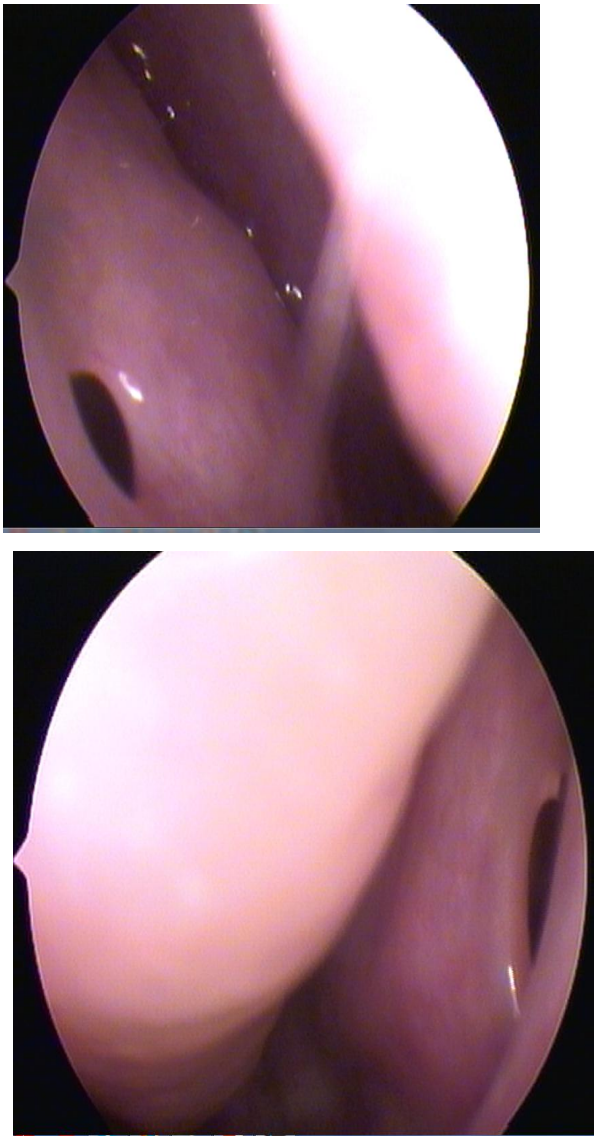


Figure 11 Bilateral AO (PNF in the a 16 year old male with CRS

In a study by Mladina et al , **68.3 %** AO were **bilateral** in patients with CRS as opposed to none the normal subjects.[17]

In another study by Mladina , Skitarelic and Casale on patients with and without post nasal discharge , **57.5 %** were present

bilaterally in those with post nasal discharge as against none in the group of healthy subjects. There

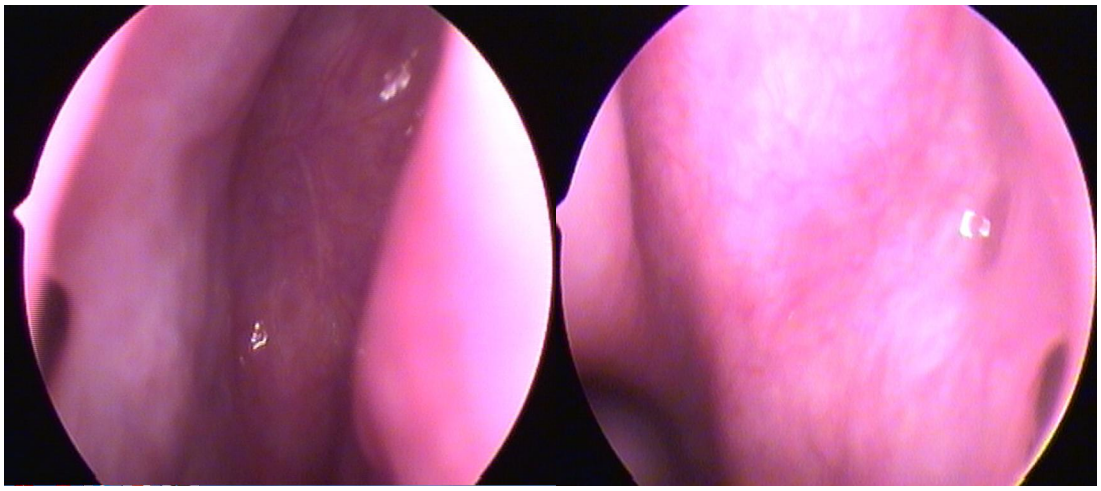


Figure 12bilateral maxillaryostia in PNF

is no mention on the right / left distribution in either of these studies.[18]Kolvekar et al[22] have reported on a 2.66% laterality.

In a cadaver study by Kumar et al the findings were as follows – right **66.7** % , left **33.3** %. There were no bilateral cases.[5]

In a cadaver study Manju et al they found that AO occurred on the Right in 60% and on the Left in 40%. [19]

A radiological study by Sheetal et al showed 13 % Right and 11 % left.

LOCATION OF ACCESSORY OSTIUM – ANF / PNF

In the group with CRS , 19 (76 %) AO were found on the posterior nasal fontanelle and 6 (24 %) in the anterior nasal fontanelle. In the unexposed individuals , all the 8 AO were found on the posterior nasal fontanelle.

PNF are larger than ANF and Accessory ostia are more commonly found on the posterior nasal fontanelle[1,2,3,8,24]. Myerson (1932) says AO commonly lies in the posterior nasal fontanelle below and behind the natural ostium. In 23 % it they lie further posteriorly, in 14 % inferiorly and 11 % superiorly.The findings in this study corroborates the literature. Other studies - Mladina et al (pnf - 19.3 %, anf–0.61 % in CRS and pnf – 0.48 %, anf – nil in healthy subjects), But a few studies which contradict it was also found – Kumar et al (cadaver study: pnf - 22.2 %; anf – 66.7 %), Manju et al (pnf – 25 %, anf – 70 %), Manjula Patil et al (pnf – 45.5 %, anf – 54.55%).

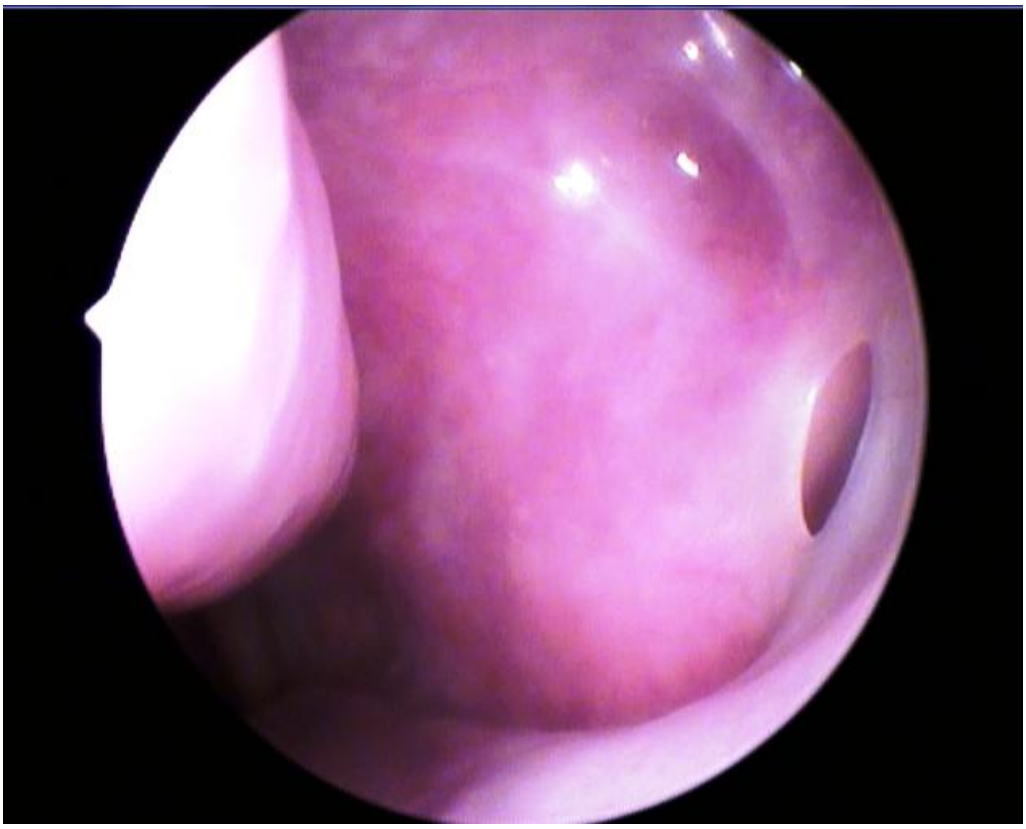


Figure 13AO in PNF

NUMBER OF OSTIA

Out of 25 AO in the CRS group 23 (92%) were single ostia and only 2 (8 %) cases had double ostia. In the unexposed group none had double ostia. Accessory ostia are usually single and occasionally multiple. Levine .H however has commented that one to three accessory ostia may be present , mostly in the anteroinferior fonatanelle.[25]

Figure 14 Double Accessory Ostia

Kumar et al have reported a 44.4 % incidence of double AO in their cadaver study. Manju Singhal et al have reported a 35 % incidence of double AO , and all of them were in the anterior nasal fontanelle. [19]

Manjula Patil et al in a cadaver study found 18 % double, 9 % multiple and 72.2 % single ostia.

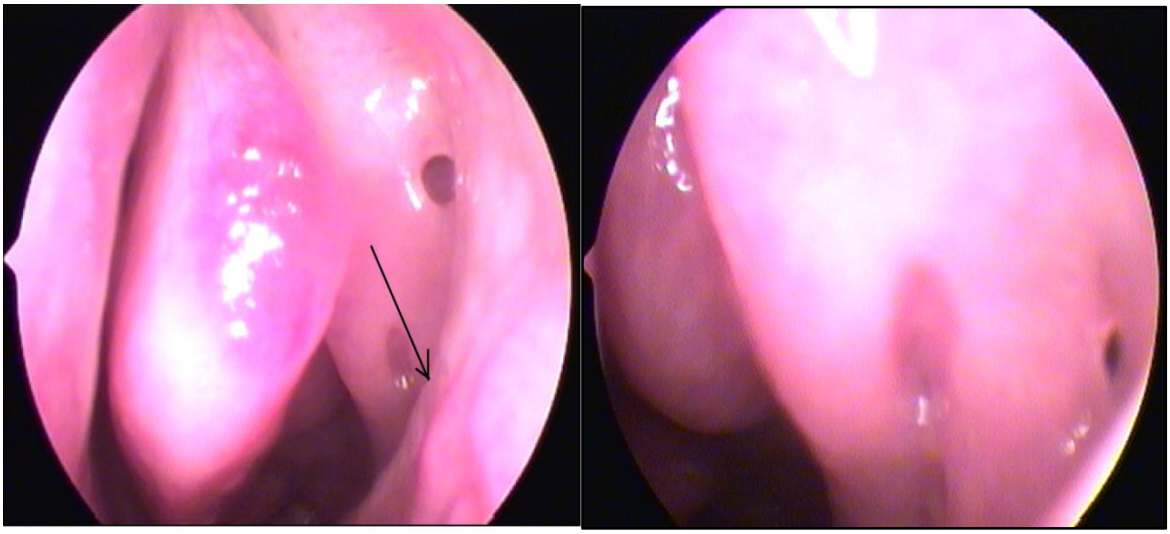


Figure 15 Lower ostia was not visualized initially as it was almost obscured by the uncinate

Again all these were cadaver studies. Of all the studies , cadaver studies have given a larger incidence of accessory ostia and here in particular the incidence of multiple / double ostia are also larger in these studies. This could be due to the fact that moist nasal mucosa undergo shrinkage after death , and following drying and fixing the fontanelles undergo damage resulting in the formation of accessory ostia.[5]

RECIRCULATION

Recirculation was found only in 3 (12 %) cases of AO in this study. All the three cases had post nasal drip and also purulence on examination.

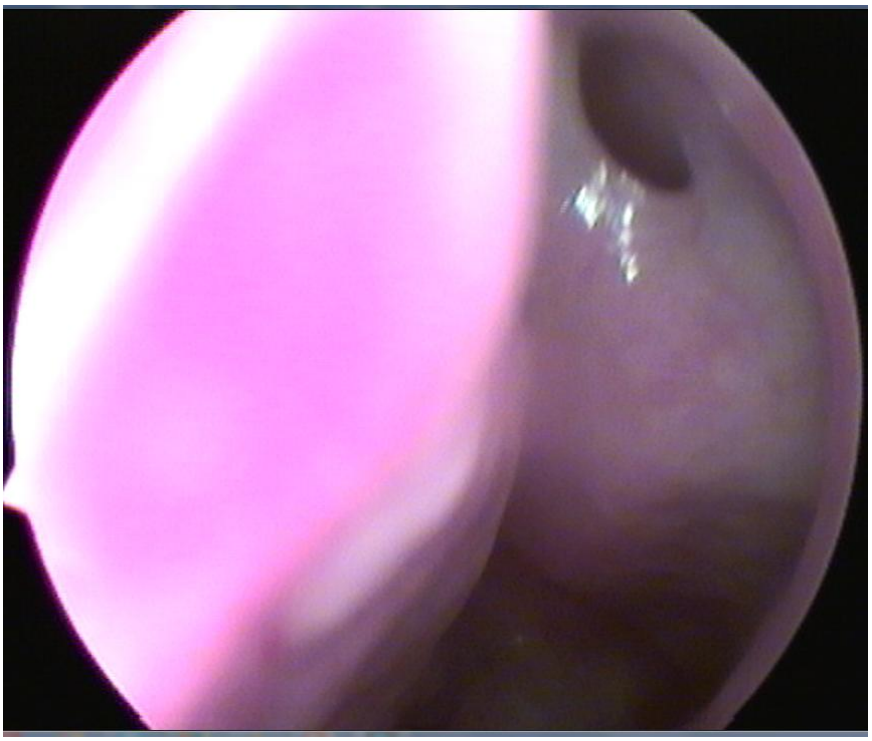


Figure 16 mucous moving out of AO

As shown in the figure below, the mucous that exits into the middle meatus re enters the maxillary sinus through the accessory ostium, taking along with it the pathogens from the nasal cavity. As the cilia beat towards natural ostium , the secretion again moves up

towards it, only to repeat this vicious cycle. This results in persistence of sinus infection.

In the figure above, secretion appears to be moving out of the accessory ostium into the middle meatus. In the normal course, though an accessory ostium is more advantageously placed than the natural one, it does not take part in the physiological drainage of the maxillary sinus secretions.

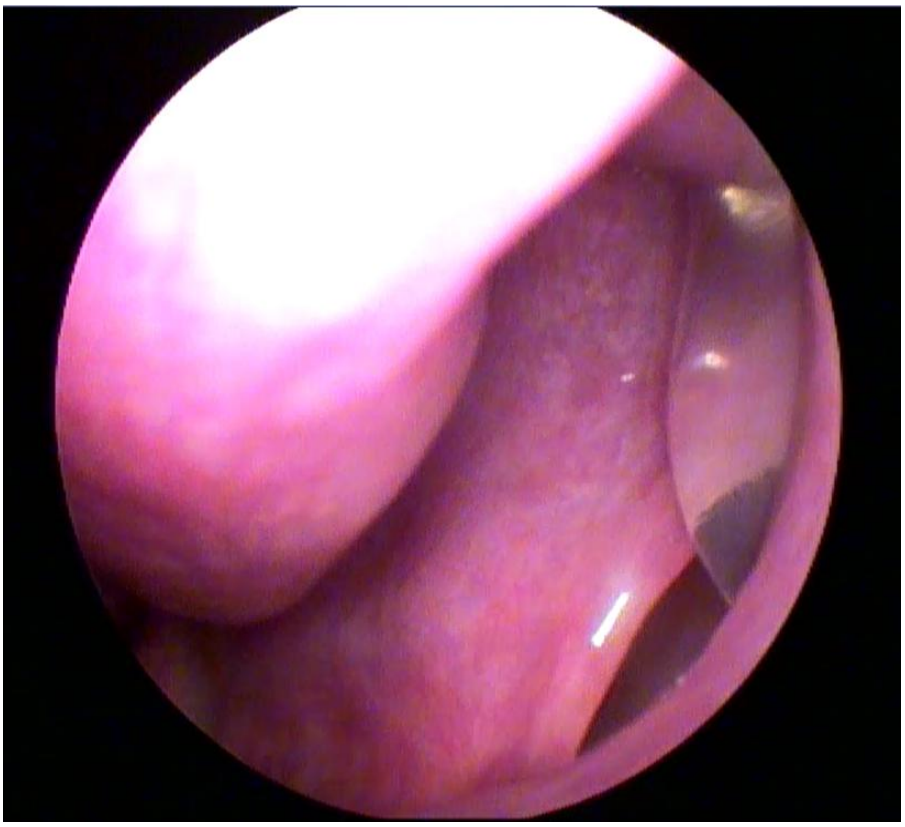


Figure 17 Circular phenomenon – the mucous secretion re entering the maxillary sinus via the Accessory Ostium

Only in the presence of any blockade or obstruction of the natural ostium does secretions get transported out through the accessory ostium, under the effect of gravity.

ANALYSIS OF THE SYMPTOMS AND SIGNS

95 % of CRS patients complained of headache and 42 % had facial pain. 80 out of 83 (96.4 %) patients complained of post nasal discharge. Hence there was no significant difference in its presence between those with and without AO. Halitosis was a complaint only for 49.3 %. However it was higher in those with AO (64 %) than those without AO (41.38 %). Purulence on examination was slightly higher in the presence of an AO (76%) in contrast to those without (72.41 %).But both these associations could not be proved statistically as the p value was > 0.05 for both.

CHRONIC RHINOSINUSITIS[9],[12]

Chronic rhinosinusitis is one of the most common otorhinolaryngologic problems affecting 32 million adults, or 16.3 % of the adult population.[9]

Histopathologically, sinusitis can be defined as the inflammation of nasal and paranasal mucosa. The underlying bone can undergo

osteitic changes. Since the mucosal lining is contiguous between the nose and sinuses, one can't spared if the other is involved. Hence the term Sinusitis has now been expanded into Rhinosinusitis.

There are two categories of changes in Chronic Rhinosinusitis

1. Polypoid mucosal changes with eosinophilia (which causes more damage to the nasal mucosa)

2. Submucosal serous gland hyperplasia.

The Rhinosinusitis Task Force of the American Academy of Otolaryngology Head and Neck Surgery have developed a classification.[12]

CLASSIFICATION	DURATION
ACUTE RHINOSINUSITIS (ARS)	7 days to ≤ 4 weeks
SUB ACUTE RHINOSINUSITIS	4 weeks to 12 weeks
RECURRENT ACUTE RHINOSINUSITIS	≥ 4 episodes of ARS / year
CHORONIC RHINOSINUSITIS (CRS)	≥ 12 weeks
Acute exacerbation of Chronic Rhinosinusitis	Sudden worsening of CRS with return to baseline.

The 1997 Task Force of Rhinosinusitis gave a consensus report on standardizing the criteria for diagnosing Chronic sinusitis. Symptoms and signs comprise the cornerstone for this criteria. The criteria have been divided into major and minor.

SIGNS AND SYMPTOMS ASSOCIATED WITH DIAGNOSIS OF RHINOSINUSITIS (1997 RHINOSINUSITIS TASK FORCE)

Major Factors	Minor Factors
Facial pain/pressure[a]	Headache
Nasal obstruction	Fever (all nonacute)
Nasal discharge/discolored postnasal drip	Halitosis
	Dental pain
Hyposmia/anosmia	Fatigue
Purulence in examination	Cough
Fever (acute only)[b]	Ear pain/pressure/fullness

[a]Facial pain/pressure alone does not constitute a suggestive history for diagnosis in the absence of another major symptom or sign.
[b]Fever in acute sinusitis alone does not constitute a suggestive history for diagnosis in the absence of another major symptom or sign.

The 1997 Task Force of Rhinosinusitis report also described the following physical findings which they divided into 2 groups. These are important but not required as a part of TFR criteria. They are -

1. those findings that are accessible to all clinicians (examination of face and anterior rhinoscopic findings)
2. those accessible only to specialists (nasal endoscopy)

The specificity of endoscopy is 85 %.

External Findings	**Anterior Rhinoscopy**	**Nasal Endoscopy**
Swelling and erythema of maxillary, frontal, ocular, orbital areas	Hyperemia	Blue discoloration of turbinates
	Edema	OMC / ostia purulence
	Crusts	Polyp
	Purulence	Septal deviation
	Polyp	Concha Bullosa
	Changes in symptoms after topical decongestion	Paradoxical Middle Turbinate
		Obstructive anomalies

The Chronic Rhinosinusitis Task Force has published another set of guidelines for diagnosing adult Chronic Rhinosinusitis where it recommends to continue the use of 1997 TFR CRS symptoms and to add the existence of physical findings – **Polyps , Purulence , Polypoid** changes [9]

[13]There is another staging system which can be considered the most accepted CT staging system called the Lund-Mackay CT staging system. It is a very simple system and has a high degree of interobserver and intraobserver agreement. It is the only system recommended by the Task Force on Rhinosinusitis for outcomes research.

Scoring is based entirely on CT findings. Each sinus is given a of 0/1/ 2:

0 = no opacification,

1 = partial opacification,

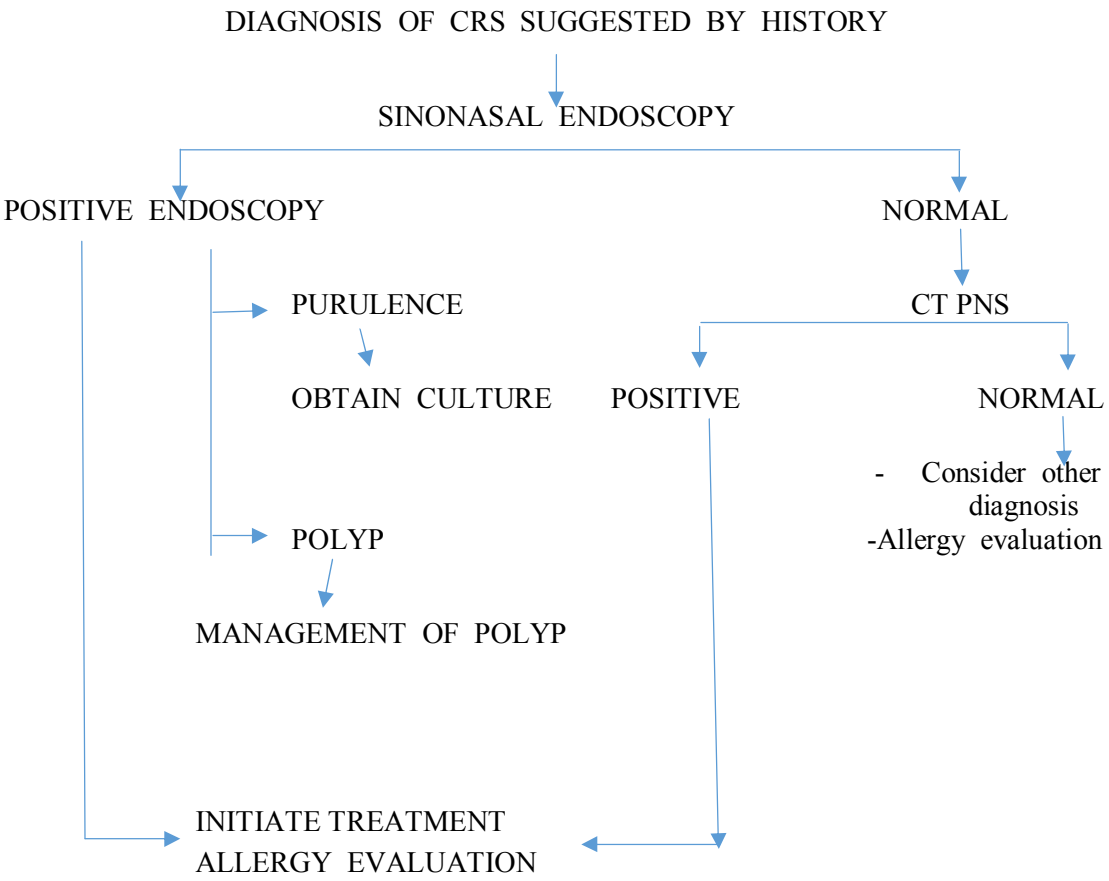
2 = total opacification .

There is a separate grading for frontal, maxillary, anterior ethmoid, posterior ethmoid, and sphenoid sinuses. The ostiomeatal complex is also included in the score. The total possible score is 24.

LUND-MACKAY COMPUTED TOMOGRAPHY STAGING SYSTEM

		No abnormality	**Partial opacification**	**Total opacification**
Anterior ethmoid	L	0	1	2
	R	0	1	2
Posterior ethmoid	L	0	1	2
	R	0	1	2
Maxillary	L	0	1	2
	R	0	1	2
Frontal	L	0	1	2
	R	0	1	2
Sphenoid	L	0	1	2
	R	0	1	2
		Non obstructed		**Obstructed**
Osteomeatal complex	R	0		2
	L	0		2

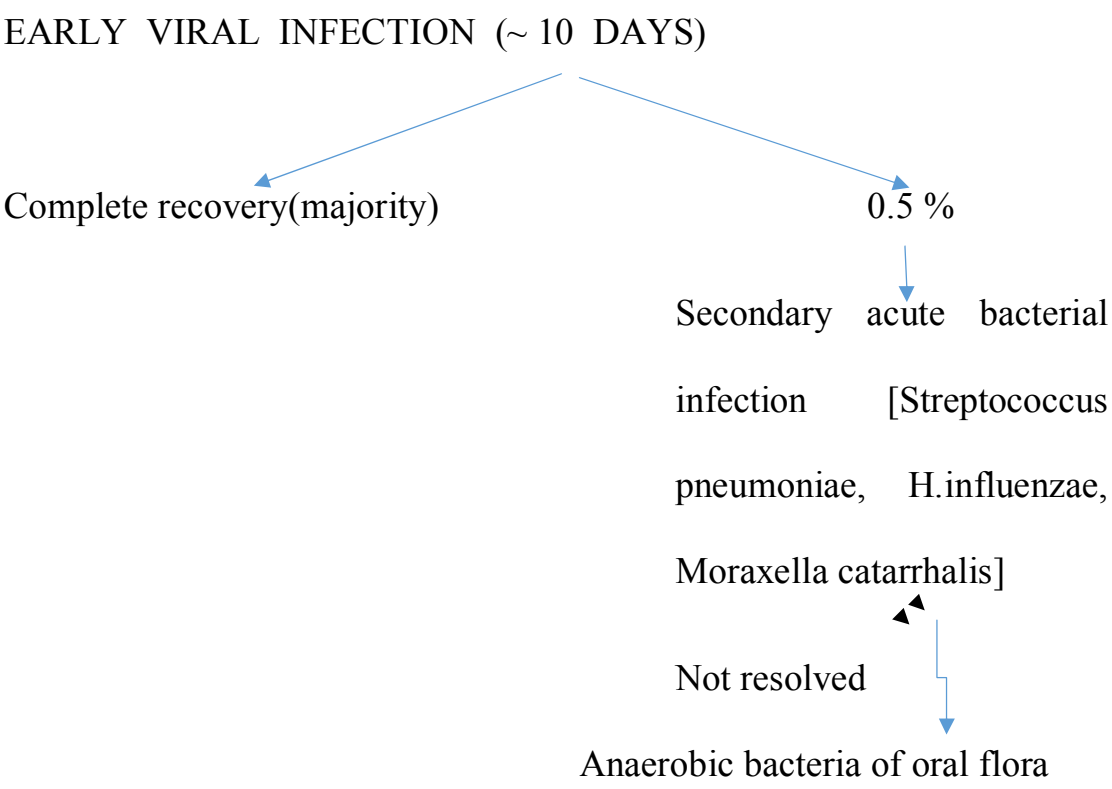
MANAGEMENT ALGORITHM FOR CHRONIC SINUSITIS[9]



TREATMENT OF CHRONIC RHINOSINUSITIS[14]

Our upper respiratory tract which includes the nasopharynx is a storehouse for pathogenic bacteria which cause respiratory tract infections including rhinosinusitis.

An Upper Respiratory Tract Infection has several phases - An initial viral infection which lasts about 10 days undergoes complete recovery in the majority. A minority develop an acute secondary bacterial infection by facultative aerobic bacteria. This if not resolved pave way for anaerobic bacteria of the oral flora.



MICROBIOLOGY OF ACUTE SINUSITIS[14]

The community acquired acute purulent maxillary, frontal and ethmoid sinusitis are caused by Streptococcus pneumoniae, Hemophilus influenzae, Moraxella catarrhalis, beta hemolytic streptococci.

Staphylococcus aureus and Hemophilus influenza are the causative agents in acute sphenoid sinusitis.

In nosocomial infections , pseudomonas and gram negative rods are the culprits.

Whereas fungal sinusitis is seen in the immunocompromised and diabetics.

MICROBIOLOGY OF CHRONIC SINUSITIS[14]

Anaerobes like Prevotella , Fusobacterium , Peptostreptococcus and aerobes like Staphylococcus aureus , Moraxella catarrhalis and Hemophilus sp. are the pathogens in chronic sinusitis.

Of these, Staphylococcus aureus, Hemophilus, Prevotella and Fusobacterium affect more than one third patients.

Pseudomonas aeroginosa and gram negative aerobic bacilli are seen in sinus infections following sinus surgeries.

In chronic sinusitis, Polymicrobial infection is more common. Hence it is more difficult to eradicate with narrow spectrum antibiotics.

In chronicity, aerobes and facultative species are replaced by anaerobes.

This is thought to be due to –

1. Selective pressure of antibiotics that enable resistant organisms to survive.

2. Persistent edema results in decreased blood supply ; consumption of oxygen by aerobic bacteria leads to decreased oxygen tension. There develops an increased acidity in the sinuses as well. These conditions are favorable for anaerobic growth.

TREATMENT OF CHRONIC SINUSITIS[14]

MEDICAL MANAGEMENT

Antibiotics should be effective against both aerobes and anaerobic Beta Lactamase Producing Bacteria. The following antibiotics can be used for treatment.-

Oral & Parenteral Forms	Only Parenteral
Amoxicillin Clavunate	Cefoxitin
Clindamycin	Cefotetan
Chloramphenicol	Cefmetazole
Macrolide + Metronidazole	Imipenem
Newer generation - Trovafloxacin	

Along with antibiotics, topical steroid sprays, nasal saline irrigation and mucolytics is also useful.[15]

In infections by aerobic gram negative bacteria like pseudomonas aeroginosa

1. parenteral aminoglycosides
2. 4^{th} generation cephalosporins – Cefipime , Ceftazidime

3. Fluoroquinolones (oral / parenteral) in post pubertal age groups.

These antibiotics should be given for 21 days. The treatment may be extended up to 10 weeks.

When a patient does not respond to medical management , surgical drainage should be done. Antibiotic treatment alone without surgical drainage of pus may not result in eradication of disease.

The reason for failure of medical treatment in chronic sinusitis may be due to the fact that chronically inflamed mucosa with a lesser blood supply is a poor medium of transport of antimicrobial agents to the affected tissue even in the presence of a therapeutic blood level. Also the reduced oxygen tension and acidic pH of the inflamed sinuses interfere with antimicrobial activity of the drugs.

[16]Another reason for drug resistance in chronic sinusitis are supposedly the formation of *Biofilms*. Biofilm is a structured community of cells , enclosed in a self produced polymeric matrix and adherent to an inert or living surface. It may be made of bacterial or fungal cells that communicate with each other in a

cooperative manner. The matrix is slime like made of polysaccharide, nucleic acids and proteins.

In a study on New Zealand white rabbits, sinusitis was induced by pseudomonas aeroginosa. On days 1, 5, 10, 20 – pus was cultured to get pseudomonas aeroginosa. The mucosa on scanning under a microscope revealed growth and biofilm.

Another study was conducted on the sinonasal specimens from patients undergoing revision sinus surgery or office based debridement. All were antibiotic non responders. Their specimens also showed findings consistent with presence of biofilms.

SURGICAL MANAGEMENT

INDICATIONS[42]

Absolute Indications

1. complications of sinusitis
2. expansile mucoceles
3. allergic / invasive fungal sinusitis
4. suspected neoplasia

Relative Indications

1. Symptomatic nasal polyps unresponsive to medical therapy

2. Chronic / Recurrent acute sinusitis unresponsive to medical therapy

Functional Endoscopic Sinus Surgery has shown to be the approach of choice in chronic sinusitis. Though the association between anatomical variations and recurrence of disease is still under dispute , surgery is acceptable when the site of obstruction complements the area of recurrent symptomatology.

When a patient complains of pain , one should keep in mind that the nasal mucosa is more sensitive to pain caused by a blocked ostium than a thickened mucosa[42]. Its easier to surgically intervene in patients who have recurrent or persistent symptoms , or those with imminent complications than those with minor sinus disease of doubtful significance. Nasal obstruction , congestion and post nasal discharge are indicative of uncomplicated sinus disease whereas severe pain associated with pressure changes like an air travel points to obstruction of the sinus[42].

PREOPERATIVE EVALUATION

Prior to surgery, a patient requires culture directed antibiotics which according to the severity of the disease may be given for up to 2 or more weeks. Steroids may put into use in case of polyposis or hyperactive mucosa. A preoperative course of 20 – 30 mg prednisone for 3 – 10 days will suffice.

Before surgery, the patient should undergo diagnostic nasal endoscopy to review the anatomy and pathology, to rule out any acute infection and to take cultures for intraoperative and postoperative antibiotic selection. [42]

Preoperative CT evaluation of each anatomic site for any variation should be done closely. The areas to stress are - the skull base , the medial orbital wall, ethmoid vessels, posterior ethmoid, maxillary sinus medial wall, sphenoid sinus, frontal recess and frontal sinus [42].

Extent of Surgery [42]

Mucosal preservation is the norm in Functional Endoscopic Sinus Surgery. It should always be borne in mind that unnecessary stripping of mucosa will lead to exposure of bone and that

denuded bone leads to delayed healing. Ciliary action may not come back to normal in those sites where the bone remains bare for more than 6 months.

The surgery should extend one stage beyond the disease established by the Computed Tomogram , or that identified during surgery. It has been shown that the underlying bone is also involved in Chronic sinusitis , and that removal of the mucosa alone does not solve the problem unless the osteitic bone is removed. This is particularly important in areas like the uncinate which is more severely involved.

ANTROSTOMY [42]

Theoretically speaking the size of the antrostomy is to be kept small. This is to protect it from over ventilation and its consequences, viz., nitrous oxide washout , slowing of ciliary activity and decreased bacteriostatic activities.

The uncinate bone occupies a considerable part of the medial wall of the maxillary sinus and is commonly affected by osteitic changes. When a diseased uncinate is incompletely removed, disease persists and scarring in the region also occurs. So in minimal

disease , a small opening of the ostium is all that is necessary. Whereas in case of a long standing and diffuse chronic sinusitis , with evidence of osteitis on CT scan or during surgery, a complete removal of the uncinate and a wide middle meatal antrostomy is advisable. If the medial wall of the maxillary sinus behind the antrostomy is displaced into the nasal airflow because of a medially extending maxillary sinus , air will directed into the maxillary sinus during inspiration. To avoid this , the medially plasced wall is to be removed up to the pterygoid plate.

What is to be done in the presence of an accessory ostium will be discussed in the coming section.

More important than the size of the antrostomy is its continuity with the natural ostium , lest recirculation of mucous and persistent infection despite surgery will result. Alternatively if the normal ostium remains closed with an open neo ostium , disease will remain in the periosteal area associated with pooling of secretions and resultant infection as the cilia only beat towards the natural ostium.

Rest of the steps of FESS are not being discussed here as the discussion is being limited to Chronic Maxillary sinusitis.

COMPLICATIONS OF ENDOSCOPIC ANTROSTOMY[42]

Complications are fortunately rare in middle meatal antrostomies. If present they are -

1. Bleeding
2. Facial pain
3. Numbness (injury to alveolar nerves supplying the meatal wall of maxillary sinus)
4. Nasolacrimal duct injury / epiphora
5. Synechiae
6. Blindness(possible, but usually associated with ethmoidectomy)

MAXILLARY SINUSITIS& ACCESSORY OSTIUM

Maxillary sinus is the largest of all human sinuses. As a result of evolution when man attained an erect posture, the primary maxillary ostium ended up being located at a higher level resulting

in the maxillary sinus draining against gravity. Maxillary sinusitis is the result of non dependent drainage and impedance of mucociliary action. The natural ostium also opens in an angle to the coronal plane.

Sinus disease and Accessory Ostium – How are they related ?

If in the presence of an intact uncinate process , you are able to see an opening which takes you into the maxillary antrum, it is rather an Accessory Ostium than a natural ostium.[32,37]Accessory ostium is also known as **Giralde's orifice**[43]. They are found over the weak areas on the lateral nasal wall called *fontanelles* which are devoid of bone and covered only by mucosal membrane of the nasal cavity on one side and that of the maxillary sinus on the other side with intervening connective tissue.

It may be assumed that

1. Sinus infections damage fontanelles. Here the formation of an accessory ostium can be likened to a tympanic membrane perforation. When pus collects in the maxillary sinus it finds a way out by perforating through the potential weak areas called fontanelles on the lateral wall of nose. It happens more

commonly through the larger posterior fontanelle, which results in the formation of AO. This is the ***Acquired Development Hypothesis*** of accessory ostium.[6]

2. AO may be a cause of sinus disease by causing disturbance in the mucociliary transport and also by allowing pathogens to easily enter the maxillary sinus.

3. If looked at this way , it can also be said that an acute nasal or sinus infection damages the fontanelles which do not have a bony component , thus creating an AO , which in turn leads to recurrent sinusitis.

Mucous clearance in maxillary sinus is exclusively by Mucociliary action. It happens against gravity. The cilia in the sinus beat only towards the Primary maxillary ostium. Though an Accessory maxillary ostium is located in a more advantageous position with respect to gravity , the secretions are not transported out through it as one might assume.

In accessory ostia of up to 4 mm, the secretions with a normal viscosity bypass around[1,31], rather than pass through it. If a larger accessory ostium is present, the part of the mucous carpet moving

through the center of the accessory ostium alone gets transported out into the middle meatus. The portion of the mucous in the periphery passes along its margins are taken into the natural ostium. On the other hand the secretion that has already been transported out through the natural ostium can reenter through the accessory ostium , and this time when it enters the mucous carries with it all the pathogens that have got adhered to its outer viscous layer. Thus the pathogens in the nasal cavity gain free access into the antrum via this 'extra hole'. This recirculation can go on and on , thus perpetuating the infection and working as a vicious cycle.

[6,26]A similar scenario can happen following endoscopic sinus surgery for chronic sinusitis. While doing a middle meatal antrostomy, if the surgically created opening is not connected to the natural ostium, it is equivalent to an iatrogenic accessory ostium. This is called the '***Missed Ostium Sequence***'[30,32]. Here the due to the same recirculation phenomenon, a persistent sinusitis results despite the surgery. Joining the surgically created ostium with the natural ostium solves the problem.

Shaffer, Ramadan et al conducted a study on chronic sinusitis patients with and without accessory ostium. They studied the ciliary area on maxillary sinus biopsy specimens taken during endoscopic sinus surgery. Electron microscopic examination of the specimens showed a significant difference in the ciliary area in the two groups. Those with accessory ostium showed a reduced ciliary area than those without it.

APPLICATIONS OF THE AWARENESS OF ACCESSORY OSTIUM

Why is the awareness about accessory ostium important ?

In today's scenario where CT scan nasal endoscopy play a major role in the diagnosis and treatment of sinusitis, it is advisable to be aware of all the anatomical and pathological variants of the nasal cavity and paranasal sinuses, one such being, our Accessory ostium.

The presence of an accessory ostium in a symptomatic individual can be pointer that he is suffering from Chronic sinusitis or say chronic maxillary sinusitis. Often you can even witness the mucous reentry during a diagnostic nasal endoscopic session. Thus

it can aid in your decision making and also in planning the surgery.

The radiologist should be aware of this condition as in a CT of the paranasal sinuses, it will as an additional communication between the maxillary sinus and the nasal cavity. sometimes when stacked one above the other, it may appear as two openings in a coronal film.

Van Alyea (1936) stated that in 20 % cases , the primary maxillary ostium is unapproachable due to the altered configuration of the uncinate or bulla , or due to the size of the ostium. In such cases when you fail to canulate the primary maxillary ostium , the accessory ostium can be used to irrigate the maxillary sinus (Levine et al , 1993). Or one can even use the fontanelles to create an alternate pathway to reestablish the ventilation.

There is a recent new invention called the 'Nasal Ventilation System'[41] which is an apparatus that is used for making an accessory ostium on the lateral nasal wall that can be used for purposes like irrigation, ventilation and drug delivery. It consists of an introducer, a ventilation tube, an irrigation catheter, a secondary

irrigation catheter, balloon catheter. The introducer with the ventilation tube and grommet is used to create an accessory ostium and then gain access through it. The irrigation catheter is channeled through the ventilation tube which irrigates the sinus cavity. it can also be used to express out the sinus contents. A secondary irrigation catheter or a balloon catheter may be introduced through the outer irrigation catheter to reach the deeper parts of the sinus. Moreover this apparatus can be used to deliver topical fluids , anti-inflammatory drugs, drug delivery substances, drug impregnated coils , foams or beads.

Now that we know the importance of an accessory ostium , lets find out how to identify it.

Distinguishing features between AMO and PMO[7,36]

Following are the differences between an AMO and PMO –

1. PMO lies in a more oblique plane. The anterior lip of PMO lies more lateral than the posterior lip. Whereas the AMO lies in the same (sagittal) plane as the lateral nasal wall.

2. PMO is ovoid in shape whereas AMO is circular.

3. In the fontanelles , PMO tends to lie anterior and / or superior to AMO.

4. PMO is always present whereas AMO is present only in a certain percentage of the population , a figure which shows a wide variation between 2 – 44 %.

5. PMO is quite difficult to see clinically , whereas an AMO is easily seen if present.

ENDOSCOPIC SINUS SURGERY AND ACCESSORY OSTIUM

When a patient is taken up for Endoscopic Sinus Surgery , a Middle Meatal Antrostomy (MMA) is not always required if the natural ostium is found patent after uncinectomy. If however an accessory ostium is found , it has to be joined to the natural ostium.[27,28,29,30,34,38] It can be done by introducing a back – biting forceps into the accessory ostium and extending it anteriorly into the natural ostium[28]. Failure to identify the natural ostium , and therefore connecting it to the accessory ostium will result in a circular flow of mucous, and persistence of infection[27,29,30]. In other words , a *Pseudo Middle Meatal Antrostomy*[36] is created with a resultant failure of Functional Endoscopic Sinus Surgery[29].

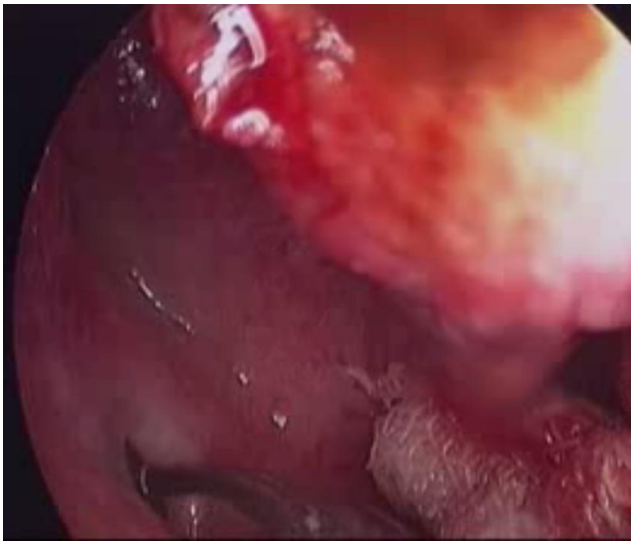


Figure 18 a posterior fontanelle AO encountered during FESS being shown using a Ball probe

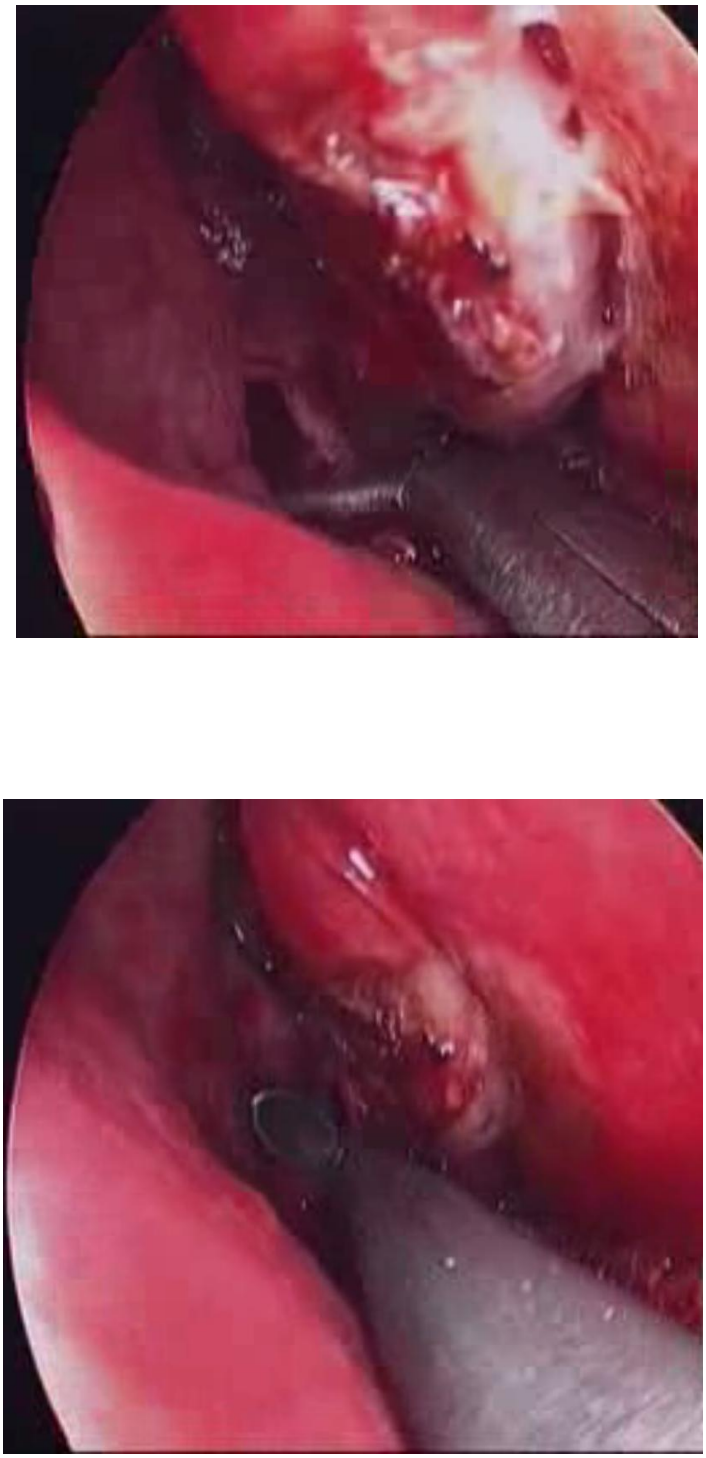


Figure 19 AO being widened and backbiting forceps being introduced into it

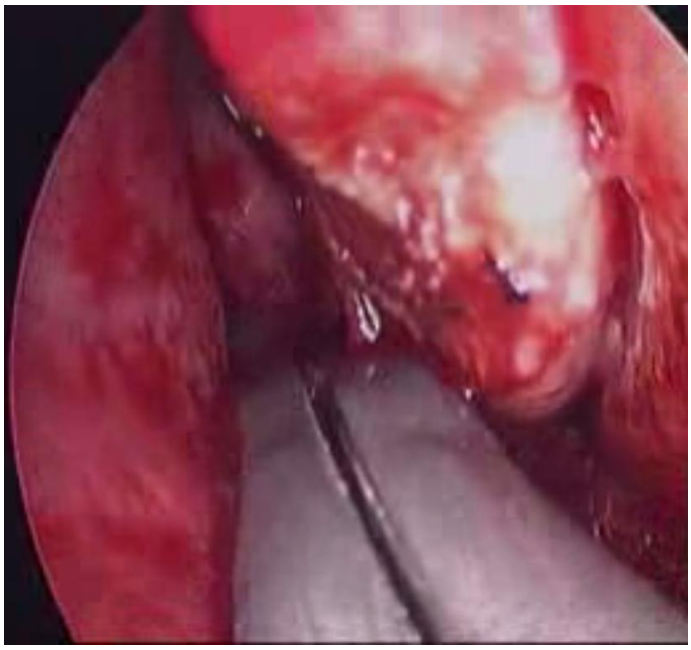


Figure 20 AO being joined to natural ostium by cutting anteriorly using Back biting forceps.

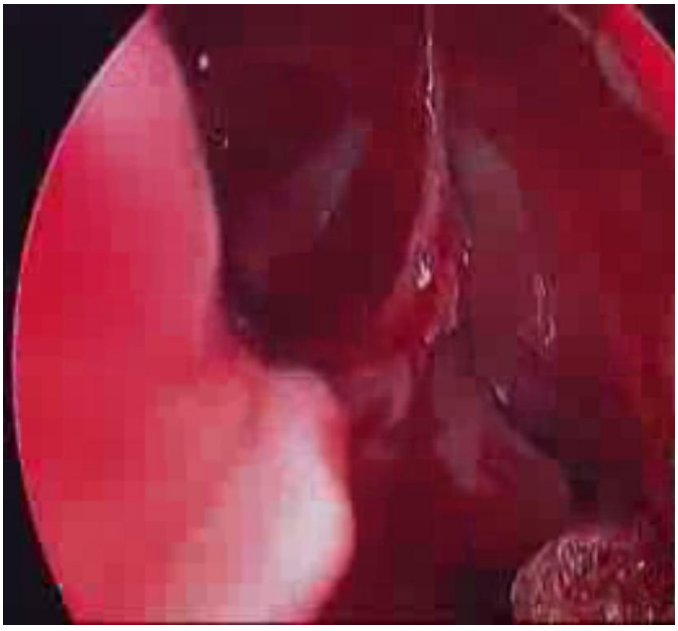


Figure 21A wide Middle MeatalAntrostomy after joining the natural and accessory ostia

While doing a middle meatal antrostomy , the ostium can be enlarged into either fontanelle. But a thing to be kept in mind is that any damage especially over the posterior ostial edge will result in disruption of mucociliary clearance [27] .

If the ostium is not found , one may perforate the posterior fontanelle just above the attachment of the inferior turbinate. In this scenario if one fails to join it to the natural ostium recirculation will result , as mentioned earlier[27]. Hence the continuity with the natural ostium may be confirmed using a 45^0 or 70^0 endoscope. But Kennedy contradicts this statement, when he says that in the absence of an accessory ostium, it is best not to enter the maxillary sinus through the posterior fontanelle, lest the mucosa should be invaginated into the sinus and stripped from adjacent walls[28].

Patients coming with recurrent sinusitis following endoscopic sinus surgery should undergo an endoscopic examination. After a course of medical treatment a CT scan of the paranasal sinuses should be taken and carefully examined for the presence of a separate natural ostium and an accessory ostium or a posterior fontanelle ostium. If it is positive, the patient should be taken up for a second sitting where the two ostia should be joined. As

mentioned above a back biter should be inserted into the accessory ostium and cut forwards to the natural ostium. A microdebrider can be used to trim away the excess tissue after creating this tissue edge.

Enlarging The Maxillary Ostium – Still An Enigma !

There are two schools of thought in this regard.

Earlier FESS surgeons supported the enlargement of the maxillary ostium towards the posterior fontanelle thus creating a wide Middle Meatal Antrostomy[29]. They advocate that a small neo ostium neither allows a proper visualization of the maxillary sinus , neither do they allow adequate tissue removal if required from inside the sinuses[30]. Hence a 1 x 2 cm wide neo ostium may be created at the expense of both the anterior and posterior fontanelles.

Whereas now the supporters of the idea - as the uncinate is located close to the maxillary ostium , the narrow transient space between it is prone to obstruction and that the removal of the uncinate alone is sufficient to restore a healthy sinus. But little data is available in support of either argument. [29]

Nitrous oxide is synthesized by NO synthase (NOS) in the mucosa of the sinuses. There are three types of NOS - Type I, II, III Of these, the most important in the production of Nitrous oxide in the sinuses is Type II. It is induced by bacterial infections. It stimulates ciliary motility , inhibits infections by bacteria, fungi and viruses , thereby playing an important role in the local innate defense mechanism of the sinuses. [29]

Previous gas studies by computational modelling have also proved that increased sinus ventilation in the form of a large ostium or multiple ostia is more detrimental than beneficial in terms of the increased nitrous oxide washout , mucosal drying (partly because sinuses have a lower density of mucous and serous glands as compared to the nasal cavity), reduced mucociliary activity and pathogen entry[4].

Kennedy et al have stated that the primary maxillary ostium has a dimension of 5 x 5 mm and that its significant enlargement leads to the dilution of sinus NO concentration and subsequent bacterial colonization of the sinus. [29]

In a study conducted by Wormald, he correlated the concentration of NO with that of the size of the maxillary sinus ostium. There appeared to be a significant reduction in concentration of NO in those sinuses and nasal cavities with large maxillary ostia (> 5 x 5 mm). But this study did not prove that a lower concentration of nitrous oxide predisposed to recurrent infections.

Hence this area requires more study and scientific proof.

Can an accessory ostium be closed ?

A study conducted by M.A Penttilä during the period 2011 – 2013 shows that accessory ostia can be closed ! They did the closure endoscopicaly making use of rotation flaps from the undersurface of the middle turbinate as abdominal fat grafts did not give satisfactory results.[39] The accessory ostium was denuded and the anterior end of the rotation flap was inserted into it. The posterior end remained attached to the middle turbinate itself. The procedure gave a fairly good closure rate. On follow up visits nasal endoscopic examination confirmed closure of accessory ostia

in 76 % patients and 75 % perforations with transient mild side effects.

Though the procedure has a theoretical advantage , the long term results are yet to be monitored as it is a very recent study.

8. CONCLUSION

- There have been various earlier studies on the presence of Accessory Ostium with a wide range of results right from 0 % to 44 %.

- In our study we found the prevalence of accessory ostium as 30 % in Chronic sinusitis and 10 % in those without the disease.

- In both the groups it was more on the left side and in the posterior nasal fontanelle than the anterior nasal fontanelle.

- Only 2 % of the diseased individuals had double ostia and recirculation was present only in 3 cases (12 %).

- Although majority of the patients presented with headache and facial pain irrespective of the presence of an accessory ostium, halitosis had a higher incidence in those with accessory ostium; it was not statistically significant though.

To conclude we would say that the presence of an accessory ostium can be considered as an indicator of maxillary sinus disease, along with the other criteria for chronic sinusitis and that it can help surgeons in their decision making as to whether a surgery is required or not.

9. ANNEXURES

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B. PROFORMA

NAME: **AGE**/SEX **I.P.No**:

D.O.A:

D.O.S:

DOD

CHIEF COMPLAINTS

1. Facial pain/pressure
2. Nasal obstruction
3. Nasal discharge/discolored postnasal drip
4. Hyposmia/anosmia
5. Headache
6. Fever (all nonacute)
7. Halitosis
8. Dental pain
9. Fatigue
10. Cough
11. Ear pain/pressure/fullness

H/O OF PRESENT ILLNESS

PAST HISTORY

H/o DM/ epilepsy/ PTB/ drug allergy/ bleeding diathesis /SHT/Cardiac disorder

FAMILY H/O:

PERSONAL H/O: Diet

Bladder and bowel habits

Addictions

GENERAL EXAMINATION:

Anemia	Jaundice	Cyanosis	Clubbing	Pedal edema	GLNE

CVS: RS: P/A: CNS:

VITALS:

LOCAL EXAMINATION:

NOSE:

EXTERNAL CONTOUR

DORSUM

VESTIBULE

SEPTUM

ANTERIOR RHINOSCOPY:

	Septum	Inf. Turbinate	Inf. meatus	Mid turbinate	Mid meatus
RIGHT					
LEFT					

PNS TENDERNESS :

RIGHT **LEFT**

COTTON WOOL TEST

COLD SPATULA TEST

COTTLE'S TEST

E/O THROAT:

ORAL Cavity

OROPHARYNX

IDL EXAMINATION

POST NASAL EXAMINATION

Examination of Ear:

	PRE AURICULAR AREA	PINNA	POST AURICULAR AREA	EAC	TM
RIGHT					
LEFT					

DIAGNOSTIC NASAL ENDOSCOPY

ANAESTHESIA:

TECHNIQUE:

ACCESSORY OSTIUM

AO +/-	Single/double/multiple	Side – left/ right	Location- ANF/PNF	Shape	Recirculation

C. ETHICAL COMMITTEE APPROVAL LETTER

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A study on the presence of accessory maxillary sinus ostium

Principal Investigator : Dr.Vrinda B Nair

Designation : PG in MS(ENT)

Department : Department of ENT
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 31.07.2012 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

23/7/12

MEMBER SECRETARY,
IEC, SMC, CHENNAI

DEPARTMENT OF OPHTHALMOLOGY
CHENNAI-1.
STANLEY MEDICAL COLLEGE

D. PATIENT INFORMATION SHEET

தகவல் படிவம்

தங்களுக்கு மூக்கில் உள்ள குறைபாடுகளை ஆராய்வதற்கு ம்ஹிசி பரிசோதனை செய்யப்பட உள்ளது.

இந்த பரிசோதனை மூலம் தங்களுக்கு மூக்கில் இயற்கைக்கு மாறாக குறைபாடுகள் ஏதேனும் இருப்பின் அதை கண்டறிவதை சிறப்பாக செய்யமுடியும். இந்த பரிசோதனையின் விளைவாக ஒப்பிட்ட ஆய்வு மேற்கொள்ளப்ட உள்ளது. இது குறித்த விவரங்களை ஆய்வில் பயன்படுத்த விரும்புகிறோம்.

தாங்கள் விரும்பினால் மருத்துவ ஆய்விலிருந்து எப்பொழுது வேண்டுமானாலும், விலகிக் கொள்ளலாம். எந்த சட்ட சிக்கலுக்கும் உட்படாமல் எப்பொழுது வேண்டுமானாலும் தங்கள்ஆய்விலிருந்து விலகிக் கொள்ளலாம்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களும், பரிசோதனை முடிவுகளும் தங்களின் ஒப்புதலின் மூலம் மட்டுமே மருத்துவ ஆய்வில் பயன்படுத்தப்படும்.

ஆய்வாளர் கையொப்பம் :

ஆய்வாளரின் பெயர் :

இடம் :

நாள் :

E. INFORMED CONSENT FORM

சுய ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் : **காது, மூக்கு, தொண்டை பிரிவு**
ஸ்டான்லி அரசு பொது
மருத்துவமனை மருத்துவக் கல்லூரி

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. எனது மூக்கில் உள்ள குறைபாடுகளை கண்டறிய DNE பரிசோதனை செய்யப்படுவதை தெரியப்படுத்தப்பட்டது. எனது மூக்கில் செய்த பரிசோதனை பற்றிய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. இந்த பரிசோதனை மூலம் எனது மூக்கு மற்றும் சைனஸ் - ல் உள்ள குறைபாடுகளை கண்டறியப்படுவதும் என்று விளக்கப்பட்டது.

இந்த பரிசோதனையின் விளைவுகளை ஆய்வில் பயன்படுத்தவும் தன்னிச்சையாக சம்மதிக்கிறேன்.

எந்த சட்ட சிக்கலுக்கும் உட்படாமல் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்று அறிந்து கொண்டேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளை மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும், அதை பிரசுரிக்கம் தேவைப்பட்டால் என்னையும் எனக்கு நடக்கும் பரிசோதனையை புகைப்படம் எடுக்கவும் நான் முழு மனதுடன் சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் : நாள் :

இடம் :

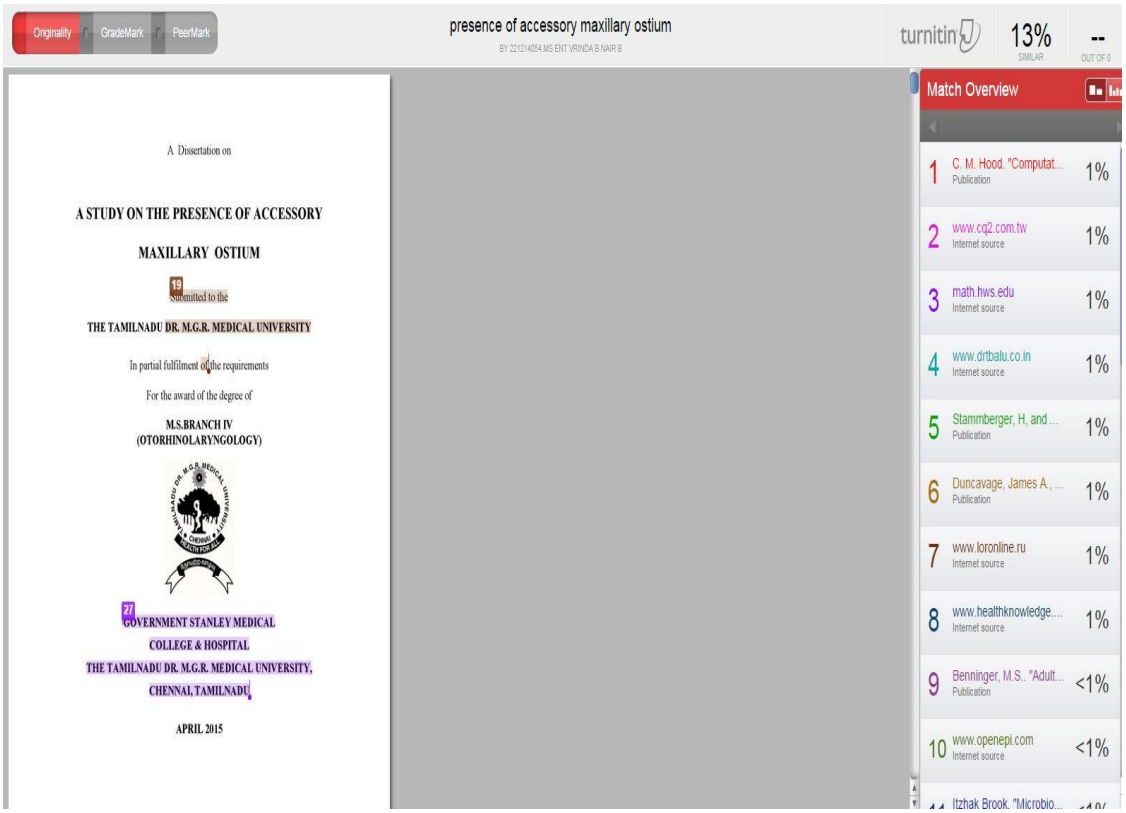
கட்டைவிரல் ஒப்பம்

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் :

ஆய்வாளரின் கையொப்பம் :

ஆய்வாளரின் பெயர் :

G. PLAGIARISM



EXPOSED

Si. No	hosp. no.	name	age	sex	duration	Fac p/pr	N. obs	Nasal dis	Hyposmia/anosmia	pnd	poe	hal	Cough	H	fever (na)	ear p/pr	dent. P	Fatigue	ton. Hyp	CSOM	max. tend.	AO	side	site	number	double	shape	recir
1	178136	prema	45	f	8 y	y	y	y	n	y	y	y	y	y	n	y	n	n		b/l	y	y	L	Pf	1	n	R	n
2	179234	renjit kumar	16	m	5 y	n	y	n	n	y	y	y	y	y	n	n	n	n	y	n	y	y	b/l	pf ,pf	1,1	n	r,r	n
3	180123	saritha	30	f	5 y	n	y	n	y	y	y	n	n	y	n	n	n	n	n	n	y	y	L	af	2	y	r,r	n
4	199873	suganta	43	f	1 y	y	n	n	n	y	n	n	n	y	n	n	n	n	n	n	y	y	r	pf	1	n	r	n
5	335122	nagamma	35	f	5 m	y	n	n	n	y	y	y	n	y	n	n	n	n	n	n	y	y	L	af	1	n	r	y
6	286906	saritha	39	f	1 y	y	n	n	n	y	n	y	n	y	n	n	n	n	n	n	y	y	b/l	af,af	1,1	n	r	n
7	287100	divya	16	f	5 y	y	y	y	y	y	y	y	y	y	y	y	n	n	n	b/l	y	y	r	pf	1	n	r	n
8	272774	sudhakar	20	m	6 y	n	y	y	n	y	n	n	n	y	n	n	n	n	n	n	n	y	l	pf	1	n	r	n
9	272895	sumati	44	f	2 y	y	n	y	n	n	n	n	y	y	n	n	n	n	n	n	y	y	r	af	1	n	r	n
10	1404126	chitra	34	f	3 y	y	n	y	y	y	y	y	y	y	n	n	n	n	n	r	y	y	r	Pf	1	n	r	n
11	346371	andal	32	f	2 y	y	y	n	y	y	n	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
12	341963	panjavarnam	41	f	1 y	n	y	y	n	y	y	y	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
13	348422	karanraj	20	m	2 y	n	y	n	n	y	y	n	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
14	369172	soundarya	18	f	1 y	n	y	y	n	n	y	y	y	y	y	n	n	n	n	n	n	n	n	n	0	n	n	n
15	341175	tamil selvi	35	f	3 y	n	y	y	y	y	y	y	y	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
16	344918	rakesh	19	m	2 y	n	y	n	n	y	y	y	y	y	y	n	n	n	n	n	y	y	b/l	pf,pf	1,1	n	r	n
17	315473	sagunthala	36	f	6 m	n	y	y	y	y	n	n	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
18	323783	rakesh sharma	26	m	2 y	n	n	y	n	y	y	n	n	y	n	n	n	n	y	n	y	n	n	n	0	n	n	n
19	322439	nirmal kumar	24	m	1 y	n	y	y	n	y	n	n	n	n	n	n	n	n	n	n	y	n	n	n	0	n	n	n
20	364446	jayanthi	40	f	1 y	n	y	n	n	y	y	n	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
21	472816	veerappan	45	m	2 y	n	y	y	n	y	y	n	y	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
22	369450	saddam hussa	18	m	1.5 y	n	y	n	n	y	y	y	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
23	376467	summayiah	23	f	4 y	n	y	n	n	y	y	n	y	y	n	n	n	n	n	n	n	y	l	pf	2	y	r	n
24	389887	sumathi	40	f	6 y	y	y	n	n	y	n	y	n	y	n	n	n	n	n	n	y	y	b/l	af,af	1,1	n	r	n
25	396811	meharunissa	30	f	7 y	n	y	n	y	y	y	n	n	y	n	n	n	n	n	n	y	y	l	Pf	1	n	r	n
26	409537	savithri	45	f	7 y	n	y	y	n	y	y	y	y	y	n	n	n	n	n	n	y	y	r	Pf	1	n	r	n
27	370756	manju	25	f	8 m	n	y	n	n	y	n	n	y	y	y	n	n	n	n	n	y	n	n	n	0	n	n	n
28	377686	murugesan	45	m	2 y	n	y	n	n	y	n	n	y	y	y	n	n	n	n	n	y	n	n	n	0	n	n	n
29	499901	srimathi	18	f	1 y	n	y	y	n	y	n	n	y	y	n	n	n	n	y	n	y	n	n	n	0	n	n	n
30	361724	malliga	42	f	2 y	y	y	n	n	y	y	n	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
31	364125	krishnakumar	31	m	5 y	n	y	y	n	y	y	n	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
32	361033	devi	32	f	3 y	y	y	n	n	y	y	y	n	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
33	371383	lakshmi	26	f	1 y	n	y	y	y	y	n	n	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
34	371890	manikandan	23	m	4 y	y	n	y	n	y	y	y	y	y	n	n	n	n	n	n	y	y	b/l	pf,pf	1,1	n	r	n
35	268712	rajesh	17	m	5 y	y	y	y	y	y	y	y	y	y	n	y	n	n	n	b/l	y	n	n	n	0	n	n	n
36	251928	priya	30	m	2 y	n	y	n	y	y	y	n	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
37	397156	kamala	28	f	1 y	y	y	n	y	y	y	y	y	y	y	y	n	n	n	n	n	n	n	n	0	n	n	n
38	394050	valli	34	f	3 y	n	y	n	n	y	y	y	y	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
39	313700	murali	20	m	5 y	y	n	y	n	y	y	n	y	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
40	384163	satyamoorthy	40	m	6 y	n	y	n	y	y	n	y	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n

Si. No	hosp. no.	name	age	sex	duration	Fac p/pr	N. obs	Nasal dis	Hyposmia/anosmia	pnd	poe	hal	Cough	H	fever (na)	ear p/pr	dent. P	Fatigue	ton. Hyp	CSOM	max. tend.	AO	side	site	number	double	shape	recir
41	394320	megalai	26	f	2 y	y	y	y	y	y	y	y	y	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
42	529390	kaja sheikh	42	m	8 y	y	y	y	y	y	n	y	y	y	y	y	n	n	n	n	y	n	n	n	0	n	n	n
43	378288	kaja moideen	28	m	6 m	n	y	n	y	y	n	y	y	y	n	y	n	n	n	n	n	n	n	n	0	n	n	n
44	394292	madhavi	24	f	6 m	n	y	n	y	y	y	n	y	y	n	y	n	n	n	n	n	n	n	n	0	n	n	n
45	407288	kathira bee	30	f	1 y	n	y	y	y	y	y	y	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
46	399391	usha	31	f	8 m	n	y	y	y	y	n	n	n	y	n	n	n	n	n	n	n	n	n	n	0	n	n	n
47	330783	shakila	30	f	1y	n	n	y	y	y	y	n	y	y	n	y	n	n	n	n	n	n	n	n	0	n	n	n
48	395195	geetha	17	f	3 y	n	y	y	y	y	y	y	y	n	y	y	n	n	y	n	y	n	n	n	0	n	n	n
49	370259	mathi	40	m	2 y	n	n	y	n	y	y	y	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
50	372700	amsa	30	f	1 y	n	y	y	n	y	y	y	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
51	394888	ravanammal	28	f	1.5y	n	y	n	n	y	y	y	y	y	n	n	n	n	n	y	y	n	n	n	0	n	n	n
52	419294	kairunisa	22	f	2y	y	y	y	y	y	y	n	y	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
53	388429	rizwana	18	f	10 m	n	y	y	n	y	y	n	y	y	n	n	n	n	y	n	y	n	n	n	0	n	n	n
54	1400562	sujatha	30	f	3y	y	y	n	y	y	y	y	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
55	1400244	ambilka	24	f	1y	n	y	n	y	y	y	n	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
56	1400168	valliyammal	36	f	5y	y	n	y	n	y	y	n	y	n	y	n	n	n	n	b/l	y	n	n	n	0	n	n	n
57	1400827	yuvarna`	20	f	2y	y	y	y	y	y	y	n	y	y	n	n	n	n	y	n	y	n	n	n	0	n	n	n
58	1401304	mujeeb	38	m	4y	y	n	n	n	y	y	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
59	1402152	pratap singh	22	m	3y	y	y	y	y	y	y	y	y	y	n	y	n	n	y	n	y	n	n	n	0	n	n	n
60	1402218	guru	36	m	8y	n	y	n	y	y	y	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
61	1402530	usman	26	m	3y	y	n	y	n	y	y	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
62	1404917	jebaraj	39	m	2y	y	y	y	y	y	y	y	y	y	y	y	n	n	n	n	y	y	r	pf	1	n	r	y
63	372089	purushothama	43	m	5y	y	y	n	y	y	y	n	n	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
64	76129	revathy	32	f	1y	n	y	n	n	y	y	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
65	11529	suganya	23	f	2y	y	y	n	n	y	y	y	y	y	y	y	n	n	n	n	y	n	n	n	0	n	n	n
66	141196	alangaram	21	f	1 y	y	Y	y	n	y	y	n	y	y	n	y	n	n	n	n	y	y	L	pf	1	n	r	y
67	154587	babu	18	m	1 y	n	y	n	n	y	y	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
68	167481	manoj	19	m	1.5y	y	y	y	n	y	y	y	y	y	n	y	n	n	n	n	y	y	r	af	1	n	r	n
69	174952	karthika	23	f	2 y	n	n	y	y	y	n	n	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
70	95263	renugadevi	20	f	10 m	y	L	n	y	n	n	n	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
71	166981	santosh	15	m	1 y	n	n	y	n	y	n	n	y	y	n	y	n	n	n	b/l	y	n	n	n	0	n	n	n
72	175139	logi	37	m	3 y	y	n	n	n	y	y	n	y	y	n	n	n	n	n	n	y	y	L	pf	1	n	r	n
73	141725	ranjith kumar	16	m	1 y	y	n	y	n	y	n	y	y	y	n	n	n	n	y	b/l	n	n	n	n	0	n	n	n
74	184597	surya	24	m	2 y	n	L	y	n	y	y	y	n	y	n	n	n	n	n	n	y	n	n	n	0	n	n	n
75	189906	nandakumar	28	m	3 y	n	r	n	n	y	y	n	y	y	n	y	n	n	n	n	y	n	n	n	0	n	n	n
76	188137	vinitha	16	f	2 y	y	n	y	n	y	y	n	n	n	n	y	n	n	n	n	y	n	n	n	0	n	n	n
77	586801	selvi	25	f	1 y	y	Y	n	y	y	y	y	y	y	n	y	n	y	n	n	y	y	r	pf	1	n	r	n
78	586811	sarath kumar	28	m	3 y	y	Y	n	y	y	y	y	y	y	n	y	n	n	n	n	y	y	r	pf	1	n	r	n
79	248380	narasimhan	26	m	5 y	n	n	y	y	y	y	y	y	y	n	n	n	n	n	n	n	y	L	pf	1	n	r	n
80	234698	malai	36	f	4 y	n	Y	n	n	y	n	y	n	y	n	n	n	n	n	n	y	y	l	pf	1	n	r	n
81	234703	jaya	41	f	2 y	n	n	y	y	y	y	y	y	y	n	y	n	n	n	n	n	n	n	n	0	n	n	n
82	228973	tamil selvan	27	m	1.5 y	y	y	n	n	y	n	y	y	y	y	n	n	n	y	n	n	n	n	n	0	n	n	n
83	273100	ghouse basha	22	m	6 y	n	n	y	n	y	y	n	y	y	n	n	n	n	n	b/l	n	y	b/l	pf,pf	1,1	n	r	n

UNEXPOSED

sl no.	hosp. no	name	age	sex	diagnosis	Fac p/pr	N. obs	Nasal dis	Hyposmia/anosmia	pnd	poe	hal	Cough	H	fever (na)	ear p/pr	dent. P	Fatigue	ton. Hyp	CSOM	max. tend.	AO	side	site	double	number	shape	recirculation
1	49790	manyan	23	M	ES	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	L	pf	n	1	r	n
2	346013	vinaya kumar	23	m	otmyc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
3	346015	valii	24	f	vn	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
4	267071	kanchana	33	f	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
5	44483	viji	26	f	vn	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
6	348411	lakshmi	44	f	SNHL	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
7	343370	bavani	40	f	mng	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
8	343394	munusamy	43	m	vc palsy	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n
9	349468	nirmala	30	f	vc cyst	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
10	450126	silendran	44	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
11	352230	hari	21	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
12	353284	kubendran	45	m	vc cyst	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
13	361931	sivaranjani	18	f	vn	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
14	361791	mohammad	24	m	otmyc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
15	46477	nagaraj	44	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
16	46980	sundaramoorth	41	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
17	358516	easa	45	m	vn	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
18	199933	rajagopal	44	m	vc palsy	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	L	pf	n	1	r	n
19	391052	venkatesh	40	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	L	pf	n	1	r	n
20	228404	tilak bahadur	45	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	L	pf	n	1	r	n
21	586819	anand	26	m	vb	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	b/l	pf,pf	n	1,1	r	n
22	586712	abirami	32	f	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	R	pf	n	1	r	n
23	294560	ragupathy	16	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	R	pf	n	1	r	n
24	29456	siva	23	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	y	R	pf	n	1	r	n
25	298370	syamala	35	f	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
26	227687	anwar	19	m	otmyc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
27	228420	devi	40	f	dcr	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
28	345342	priya	20	f	mng	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
29	25287	gangadharan	40	m	ES	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
30	38030	thasneem	36	f	ES	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
31	147643	lakshmi	46	f	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
32	27698	manikandan	23	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
33	20876	bhuvaneswari	30	f	pas	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
34	289756	sulekha	28	f	sms	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
35	28703	manikandan	17	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
36	253793	jabamalai	39	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
37	387367	thangaraj	45	m	pl.ad	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
38	128908	nazeer	33	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
39	138766	pugazanthi	30	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
40	128936	chinnadurai	43	m	MOE	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
41	146735	raziya	45	f	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
42	37698	murugesan	36	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
43	140567	shakeela	16	f	pas	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
44	38965	fiaz	40	m	sms	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n

sl no.	hosp. no	name	age	sex	diagnosis	Fac p/pr	N. obs	Nasal dis	Hyposmia/anosmia	pnd	poe	hal	Cough	H	fever (na)	ear p/pr	dent. P	Fatigue		ton. Hyp	CSOM	max. tend.	AO	side	site	double	number	shape	recirculation
45	37609	seenivasan	39	m	bppv	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
46	27462	murugan	37	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
47	37876	sukumari	40	f	osc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
48	348470	uma	28	f	kob	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
49	369150	arun	16	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
50	315489	karuna	17	f	fb throat	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
51	47278	raziya	24	f	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
52	348410	balaraman	40	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
53	369140	mohini	37	f	dcr	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
54	315389	pushpa	38	f	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
55	344900	abdul khader	39	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
56	368123	lathika	44	f	mng	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
57	272809	sagunthala	23	f	jug ect	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
58	322420	maheswari	30	f	fb ear	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
59	273110	guru	30	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
60	346350	mukesh	18	m	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
61	140478	jagadeesan	37	m	pl.ad	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
62	347136	john	35	m	val cys	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
63	261895	sampath	35	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
64	342874	jeelan	17	m	bvfp	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
65	433785	malkonadyan	45	m	fb throat	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
66	346279	silambarasan	34	m	fb throat	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
67	1E+06	ragupathy	40	m	thy fis	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
68	457482	fathima	17	f	NV	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
69	230852	kuppusamy	45	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
70	204362	chitra	38	f	mng	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
71	427584	shanti	40	f	bvfp	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
72	270283	shalini	19	f	mng	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
73	1E+06	hassan	29	m	smf	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
74	1E+06	jahangir	25	m	leu. pl	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
75	341298	syed	19	m	ran	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
76	272700	mukesh	17	m	pub.ph	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
77	163800	kaliyan	45	m	kob	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
78	145622	anandan	43	m	mng	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
79	204376	kathija bee	29	f	pap. Ca	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
80	305674	umapathy	45	m	vb	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
81	306186	malliga	43	f	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
82	2E+06	saravanan	39	m	mhc	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n
83	1E+06	sakunthala	37	f	vn	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	n	0	n	n

KEY TO MASTER CHART

y - yes

n - no

b/l - bilateral

r - round

af - anterior fontanelle

pf - posterior fontanelle

fac p/pr - facial pain/pressure

N. obs - nasal obstruction

N. dis - nasal discharge

pnd - post nasal discharge (discolored)

poe - purulence on examination

hal - halitosis

recir - recirculation

H - headache

fever (na) - fever all non acute

ear p/pr - ear pain/pressure

dent. p -dental pain

ton. hyp - tonsillar hypertrophy

CSOM - chronic suppurative otitis media

max. tend - maxillary tenderness

AO -accessory ostium

ES - eagle's syndrome

otmyc - otomycosis

vn - vocal nodule

mhc - master health check up

SNHL - sensory neural hearing loss

vc palsy - vocal cord palsy

vc cyst - vocal cord cyst

NV - normal volunteer

vb - vestibulitis

dcr - dacrocystitis

mng - multinodular goitre

pas - preauricular sinus

sms - submandibular sialadenitis

pl.ad - pleomorphic adenoma

jug ect - jugular ectasia

fb ear - foreign body ear

val cys - vallecular cyst

bvfp - bilateral vocal fold paralysis

thy fis - thyroglossal fistula

smf - sub mucous fibrosis

leu. pl - leucoplakia

ran - ranula

pub.ph - puberphonia

pap. ca - papillary carcinoma thyroid

MOE - malignant otitis externa

bppv - benign paroxysmal postional vertigo

osc - otosclerosis

kob - keratosis obturans

fb throat - foreign body throat